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THE
LARYNGOSCOPE.

VOL. LIX APRIL, 1949. No. 4

MANAGEMENT OF CARCINOMA OF THE LARYNX.*

HENRY B. ORTON, M.D.,
Newark, N. J.

During the past 50 years real progress has been made in the surgical treatment of malignant disease of the laryngopharynx. Formerly, the operative mortality varied enormously with the type of operation performed. The operative mortality in thyrotomy was 10 to 20 per cent; 26 to 35 per cent in hemilaryngectomy, and 47 to 60 per cent in laryngectomy. This high mortality rate caused Butlin, in 1887, to say that "results were so gloomy that there seemed to him to be scarcely justification for the performance of a radical operation for cancer in this area. Scarcely a single case had been cured and the mortality extremely high and success surprisingly small." Such were the results in surgery in malignant disease of the laryngopharynx at the turn of the century. Compare the above with the results at the present time of thyrotomy[1] with 85 to 95 per cent cured and total laryngectomy with 60 to 90 per cent[2] cured, depending upon the site of the lesion; with an operative mortality rate of 1.8 to 4.3 per cent.

More than 20 years ago, Sir Wilfred Trotter[3] said: "What may be our hopes about the discovery in the treatment of

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malignant disease, of methods more specific and less harsh, surgical operation must be admitted to be the most useful and trustworthy means we now possess." This statement still must stand today. The pathological and clinical principles, combined with the development of surgical technique, have extended the field to which they can be applied.

In this series of cases the histological structure of the malignant variety of cancer attacking the laryngopharynx, the squamous cell type predominates. Regarding sex, the males predominate in the ratio of 12 to 1. The youngest patient was 13½ years of age and the oldest, 76.

We are concerned with two groups of cases: first, those malignancies within the larynx proper; the epilaryngeal or extrinsic cancer of the larynx, as some may wish to term it. Second, those malignancies of the lateral and posterior wall of the pharynx, including the postcricoid area. Lately, physicians have developed an increasing interest in the treatment of this disease, yet there are some who doubt the results of treatment by surgery and turn all cases over for irradiation. This is especially true regarding the epilaryngeal type of cancers, and is causing a great deal of controversy as to their treatment. It is true that these extrinsic cancers are insiduous in their onset and spread rapidly to the lymph nodes at a very early stage. Some authorities say that patients are not arrested or cured by surgical operation, and should be turned over to the radiologist for deep X-ray therapy. Some patients believe that operation for cancer in this area has a high mortality rate, and that they will lose their voice forever. Some physicians even tell their patients that such is the case. This is not true.

As I have already stated, the operative mortality in my series is 1.8 per cent. As to regaining speech, I am convinced that a man without his larynx can talk, whistle and smoke again, or even play a musical instrument, if he has the determination. To rehabilitate the laryngectomized patients in speaking is part of our postoperative care.

Surgical removal means a procedure sufficiently radical to remove the entire primary lesion. The surgical treatment given one case must be governed by the type, extent of growth and its location. Emotions, economics or conservatism must not be considered. Conservative methods, as saving cartilage, muscles or attachments for cosmetic or functional betterment are unsurgical and unsafe in the treatment of cancer of the laryngopharynx.

Diagnosis should be made early, as the growth is readily seen by direct or indirect examination; yet it is interesting to note that 58 of the 168 cases, or 34 per cent, I found were inoperable at the time of my first examination. This is something to think about concerning early diagnosis. Twenty-one per cent needed tracheotomy on the first visit. It is true that cancer cannot be cured in all cases, but certainly if this region is examined with painstaking thoroughness much can be accomplished by earlier diagnosis and radical operation.

Fig. 1. Drawing showing the pre-epiglottic space marked X.

I[13] personally look at all biopsies with the pathologist before operating and find this cooperation most valuable. New, in his observation on the microscopic extension of the tumor taken at 5, 10 and 15 mm. from the apparent margin of the growth, found that the maximal extension of highly malignant tumors was 15 mm. This has been brought out by Broyles and Snitman, both of whom have shown that the cancer cells have extended beyond the apparent growth histologically, in the anterior commissural tumors of the cord — an additional reason why the pre-epiglottic space is invaded early, and should be removed with the larynx. I have demonstrated this invasion of the pre-epiglottic space[4] many times at operations in cancer of the epiglottis below the hyoepiglottic ligament. This pre-epiglottic space, or the space of Boyer, as it is sometimes called, was described by Jean LaRoux[5] in an article published in 1936, and at the same time Tambein demonstrated histologically that cancer of the vestibule and the ventricle of Morgagni invaded the pre-epiglottic space and stated that the classical laryngectomy is insufficient for the complete eradication of cancer in these cases.

The surgical procedures in malignancies of the laryngopharynx are *1.* thyrotomy or laryngofissure; *2.* laryngectomy; *3.* laryngopharyngectomy; *4.* lateral transthyroid pharyngotomy.[6,7] The indications and relative merits of these various operations will now be taken up, together with the different locations of these tumors.

LARYNGOFISSURE OR THYROTOMY.

When the growth is situated on one vocal cord, at the middle third with both extremities of the cord free from disease, and the cord not fixed, but retains its full mobility, thyrotomy is the choice operation. Any fixation of the cord or arytenoid shows muscular involvement, and any conservative surgery will be inadequate. In my own experience in properly selected cases for thyrotomy, the results have been 93 per cent living over five years. My first thyrotomy patient, for true cordal cancer of the larynx, is still living 30 years after operation.

TABLE 1. LARYNGOFISSURE OR THYROTOMY.

	14.		
Alive and well	9	Alive and well under 1 year	2
Died from recurrence	1	Alive and well 1 to 5 years	1
Died from other causes	4	Alive and well 10 to 30 years	6
	14		9

Successful cases, nine plus four; total, 13, or 93 per cent cures.

INTRINSIC.

Fig. 2. Intrinsic cancer (about 75 per cent of laryngeal cancer is intrinsic). (1) shows the vocal cord, a favorite site for intrinsic cancer, which spreads along the cord and may become subglottic and spread to the opposite cord or across the commissure. (2) shows the ventricular band and ventricle. These are rarely involved, but their involvement is more dangerous than that of the cord, causing early glandular involvement. (3) shows the interarytenoid area, which is rarely affected.

LARYNGECTOMY.

The indications for this operation are:

a. When the growth has extended to the anterior commissure, or has extended backwards to the arytenoid cartilage.

b. The epiglottis is involved below the level of the hyoepiglottic ligament.

c. The growth has crossed to the opposite side and there is a subglottic extension.

d. The growth springing from the ventricle or ventricular fold.

e. The growth has invaded the thyroid cartilage or the cricothyroid membrane.

f. Definite fixation of one or both vocal cords.

g. The growth is subglottic in origin.

h. In a recurrence following a laryngofissure operation.

i. Where the larynx has been subjected to unsuccessful irradiation in selected cases.

To be considered in these cases is the extent of the growth and its location. The growths are usually of squamous type and are readily seen by direct or indirect examination of the larynx. With a growth so situated as above stated, a laryngectomy is the procedure of choice.

LARYNGOPHARYNGECTOMY.

The surgical treatment for the epilaryngeal group or extrinsic type of cancer is more difficult, in that the entire larynx with some of the pharyngeal tissue must of necessity, accord-

TABLE 2. PHARYNGOTOMIES.

	17.		
Alive and well	4	Alive and well 5 to 10 years	2
Died from recurrence	6	Alive and well 10 to 15 years	2
Died from other causes	7		—
	17		4

Successful cases, four plus seven; total, 11, or 65 per cent cures.

SUBGLOTTIC.

Fig. 3. Subglottic cancer originates on the inner or the under surface of the vocal cords or the subglottic area (chiefly in the anterior half of this region). (1) shows intrinsic cancer of the cord becoming subglottic at the anterior commissure; (2) intrinsic cancer of the cord on the inner or under surface but below the free edge (invasion of the opposite cord is by the subglottic route or across the commissure); (3) the true subglottic cancer.

ing to the site of tumor, be removed along with the larynx, with the possible exception of cancer of the epiglottis, on the

laryngeal surface, above the level of the hyoepiglottic ligament. If such a case presents itself, then the upper part of the epiglottis can be removed by a lateral transthyroid pharyngotomy, with excellent prospects of a lasting cure.

Let us take up the different locations of these tumors of the epilaryngeal group and the proper procedure to carry out.

THE EPIGLOTTIS.

Some authorities say that a cancer on the edge, or on the laryngeal surface of the epiglottis, may be approached by a lateral pharyngotomy and the epiglottis excised, so as to leave the main structure of the larynx intact. I shall qualify that statement by saying that the growth on the epiglottis must be above the level of the hyoepiglottic ligament since early invasion of the pre-epiglottic space is certain to take place with any growth below this level. Suffice it to say that I strongly recommend that with any growth of the epiglottis below the level of the hyoepiglottic ligament, the epiglottis with the larynx and hyoid bone should be removed *in toto*. This seems radical, but radical we must be if success is our goal. Any growth above the level of the hyoepiglottic ligament may be removed by lateral pharyngotomy.

THE ARYEPIGLOTTIC FOLDS.

If seen very early, cancer in this region may be removed by laryngeal pharyngotomy, but usually by the time the patients present themselves for operation, the growth has already extended beyond the area for this type of operation, and we must resort to a wide excision of the larynx with the hyoid bone and pre-epiglottic space and perhaps some of the lateral wall of the pharynx.

TABLE 3. LARYNGECTOMIES AND LARYNGOPHARYNGECTOMIES.

220.

Alive and well	118	Alive and well under 1 year	21
Died within 3 wks. of operation	6	Alive and well 1 to 5 years	29
Died from recurrence	54	Alive and well 5 to 10 years	31
Died from other causes	42	Alive and well 10 yrs. and over	37
Lived under 5 years	17		
Lived over 5 years	25		
	220		118

Successful cases, 118 plus 42; total, 160, 72.7 per cent cures.

EXTRINSIC.

Fig. 4. Extrinsic cancer; in this group are included neoplasms growing from the epiglottis, the aryepiglottic folds, the arytenoid cartilages, the pyriform sinuses and the pharyngeal surface of the cricoid cartilage (post-cricoid cancer). (1) shows the margins of the epiglottis; (2) the aryepiglottic folds; (3) the arytenoid cartilages; (4) the pyriform sinuses; (5) the pharyngeal surface of the cricoid cartilage.

MIXED.

Fig. 5. In this class, a combination of extrinsic and intrinsic, must be placed a number of cases which only present themselves in an advanced stage when it is impossible to determine the site of origin. (1) shows extrinsic cancer becoming intrinsic; (2) intrinsic cancer becoming extrinsic; (3) shows subglottic cancer becoming intrinsic and breaking through the cricoid membrane, becoming extrinsic on the neck.

THE PYRIFORM SINUS.

Here the tumor infiltrates both the outer as well as the inner walls of the sinus which is so closely connected with the larynx that nothing short of a complete laryngectomy with the hyoid bone and pre-epigottic space would be of any avail. Even a node resection may be necessary in conjunction with this procedure.

THE LATERAL WALLS OF THE PHARYNX.

This type of tumor or cancer can be removed by lateral pharyngotomy, preceded by a node dissection at the time of operation, preferably, and followed by a plastic reconstruction at a later date if necessary.

THE POSTCRICOID.

If seen very early, a lateral pharyngotomy with plastic reconstruction following at a later date may be done; but if the cord is fixed, and the cancer has advanced, nothing but a laryngopharyngectomy is of any use.

The operation of lateral pharyngotomy is an ideal operation for inspection and removal of cancer of the lateral wall of the pharynx; however, it has its limitations. It is like the laryngofissure operation in intrinsic cancer of the larynx. Lateral pharyngotomy is only for very early cases of extrinsic cancer of the lateral and posterior wall of the pharynx and the epiglottis as mentioned above. Complete destruction of all cancer cells is at present the only known method of effecting a permanent cure, whether this be by surgery, irradiation or by some biological change in our makeup.

METASTATIC INVOLVEMENT.

The management of carcinoma of the larynx with metastatic involvement of the neck could mean metastatic involvement of the neck at the same time that the larynx is involved, or it could mean metastatic involvement following laryngectomy. Naturally, from the epilaryngeal and hypopharyngeal groups where the lymph drainage is free, we have, therefore,

early meastasis to the regional lymph nodes. It is well to know the anatomy of the lymphatic drainage from this area; however, we must remember that in the early cases these nodes may or may not be carcinomatous.

LYMPHATICS.

The submaxillary glands drain the side of the tongue along with other parts of the face; therefore, with any involvement of the posterior part of the tongue, from the lingual surface of the epiglottis, the submaxillary gland with its nodes should be removed.

DEEP SET OF CERVICAL GLANDS.

A. The infrahyoid glands. These lie on the thyrohyoid membrane and drain the front of the larynx. It is important to remove the pre-epiglottic space in laryngectomy.

B. The prelaryngeal glands. These lie on the cricothyroid ligament draining the larynx.

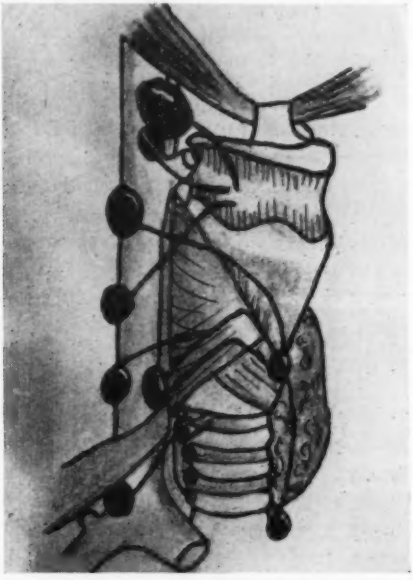

Fig. 6. Drawing showing lymphatic drainage of larynx.

Trotter has shown that their afferents pass through a small foramen in the middle of the cricothyroid ligament. In cancer of the larynx these glands are often the first to become enlarged; they also assist in drainage of the thyroid. It is wall, therefore, to remove the cricoid cartilage with the larynx. The deep cervical chain receives ultimately all the lymph from the sections above mentioned. This deep cervical chain consists of a number of large glands lying in relation to the carotid sheath. A few of these occupy an outlying position behind the pharynx. This deep chain lies along the side of the pharynx, trachea and esophagus, and extends from the base of the skull to the root of the neck. These are divided into the superior and inferior by the point of bifurcation of the common carotid or the crossing of the omohyoid muscle. Both are in close relationship with the internal jugular vein, and in removing them it is necessary to strip them from the vein, or better, to remove the jugular vein along with them. Some of the glands of the inferior group project beyond the posterior border of the sternomastoid into the posterior triangle of the neck. There are a few glands of this group that lie in the groove between the trachea and esophagus and drain the thyroid paratracheal glands. A large gland, situated below the angle of the jaw between the internal jugular and facial veins, is known as the main gland of the tonsil. Another large gland at the bifurcation of the carotid just below the cornua of the hyoid bone, is called the main gland of the tongue. Finally, a large gland on the carotid where the anterior belly of the omohyoid muscle crosses it, is known as the supra omohyoid gland.

The main gland of the tongue, in cancer of the larynx involving the base of the tongue, is the one that enlarges so rapidly postoperatively; and in many cases this gland may be removed and followed by irradiation. The dissection of the neck should take in the area from the angle of the jaw and tip of the mastoid downwards to below the posterior belly of the omohyoid muscle; removing the jugular vein, submaxillary gland, fascia, hyoid bone with the infrahyoid muscles and larynx en masse, and part of the thyroid gland if needed.

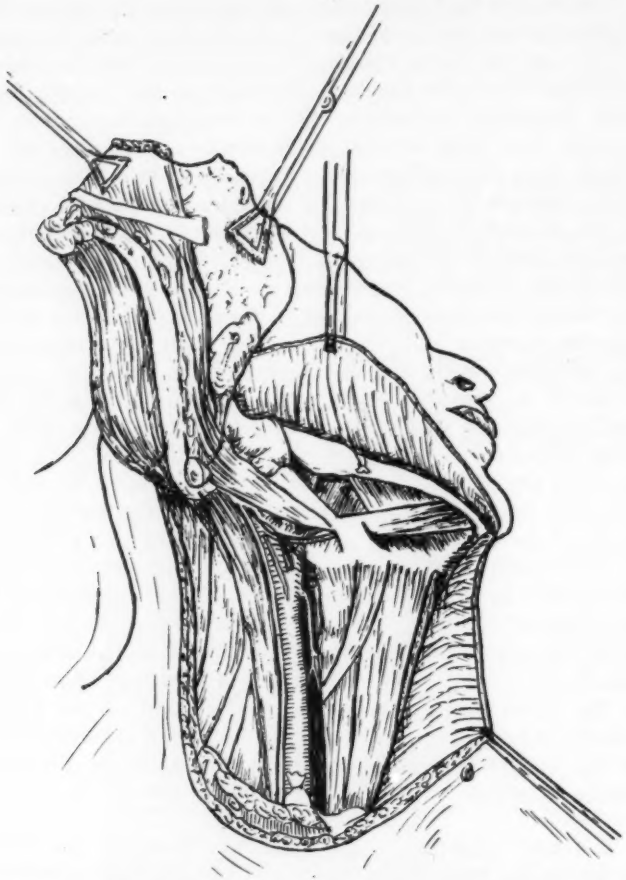

Fig. 7. Drawing of block resection of the neck, showing structures to be removed in metastasis of lymph nodes.

This is what Brunschwig[3] calls a panlaryngectomy. We must remember that the flow of lymph following a resection may have a retrograde movement and involving the glands in the postauricular area, posterior triangle of the neck ending in the retropharyngeal glands. With involvement of the base of

the tongue from the lingual portion of the epiglottis, we must remember that the cancer cells in the tongue may pass readily from one side to the other; from this it becomes necessary to deal with both sides of the neck. When we have a patient who has the entire length of the deep cervical chain of lymphatic glands involved in a fixed, hard, firm mass, extending to a point below the infrahyoid muscle, it is needless to say that we cannot expect to cure the cancer. By that time the cells have already entered the thoracic duct and into the general blood stream.

In the lateral transthyroid pharyngotomy, a block resection of glands and fascia is done before the pharynx is opened to attack the growth adjacent to the larynx.

In advanced laryngeal carcinoma with movable cervical nodes, a wide T-incision is made; the same resection of glands and fascia is done, and the larynx, hyoid bone with the infrahyoid muscles attached to the larynx and the necessary part or all of the lower pharynx is removed en masse. The raw surface is covered with skin, leaving an upper and a lower stome, which must be closed later by one or more plastic operations for the reconstruction of the pharynx and upper esophagus. These wide incisions are necessary for proper radical surgery in these cases and none should attempt to do this type of surgery by any narrow field. This wide incision gives one the extensive exposure to the area. Often nodes are seen that were not palpable preoperatively, and when found, proper care can be given them. New has also reported excellent results by the use of electrocoagulation in selected cases of advanced laryngeal and pharyngeal cancer.

In these operations we have procedures amenable to a possible cure; if not a cure, then certainly an appreciable palliative control of the disease. In these extensive laryngectomies, pharyngotomies or laryngopharyngectomies, one must have a good knowledge of the extent of the growth, made possible by X-ray and direct examination, before operation, then the type of operation decided upon and the basic steps carried out. A thorough knowledge of the anatomy of the neck is a prerequi-

site for successful surgery. One may begin with a lateral transthyroid pharyngotomy and upon opening the pharynx finds it necessary to change his operation to extensive laryngopharyngectomy, with reconstruction of the entire cervical esophagus. The surgeon must be able to meet and change his procedure as conditions present themselves during the operation. When one describes the various steps of any of these operations, only the general basic principles can be explained. The shock of these operations is not so severe as one would expect and with the use of pencillin and sulfadiazine therapy, operative mortality is reduced to almost nil.

Treatment by irradiation has been widely studied in the treatment of laryngeal and pharyngeal carcinoma and is very interesting. Unfortunately, the results of the treatment do not lend themselves to satisfactory comparison, and the subject is not sufficiently advanced to be reviewed historically. For instance, the type of case in which the radiologists promise[10] most is that type of intrinsic cancer limited to one cord. It is in this type of case that the lasting benefits of treatment by surgery are well established, and the percentage of cures by surgery far surpasses those cured by irradiation. To me, in all cases where surgery can hold out good promise of lasting cure, it still must be given the preference. When the extent or nature of the disease, or the age of the patients are contraindications, then irradiation should be given a trial. It is commonly supposed[9] that the use of some form of irradiation has greatly simplified the treatment of cancer of the larynx and pharynx. This supposition is erroneous. It has given us a new and a very powerful addition to our means of attack, but, far from simplifying the attack, it has been made more than ever dependent upon experienced judgment and expert handling. It is posible even today that total results as to cure or notable periods of freedom from recurrence are actually worse for the introduction of irradiation, since it seems likely that many patients who might have had a good chance of cure by operation in competent hands have had this chance shattered by ineffective irradiation treatment. This may be only a transit phase, for a proper appreciation of the technical requirements of sound irradiation therapy is already replacing

the cruder view and in time must result in the full benefits of irradiation being made manifest. I am absolutely in accord with Colledge when he states, "As the results of irradiation, however, are so impossible to predict and the results at the and of three to five years so poor that I have abandoned it for every operable case, and only recommend it when the operation is refused or the case is inoperable. It is then a useful palliative measure and the growth often disappears for some months, but the lasting results are few." The claims of the radiologist now complicate the picture. These claims are often exaggerated; however, they may not be without good foundation. The surgeons use a five-year period as their basis for success, whereas the radiologists do not. Most of their claims are based on the fact that the cases best suited for radiology are the very ones that are best suited for surgery, and in the inoperable cases when surgical treatment is more difficult, the radiologist has very little success. True palliative results may be obtained, but patients are often made worse by perichondritis and radionecrosis. The tables herein presented are used to substantiate the above statements.

CONCLUSIONS.

It is difficult to compare and appraise vital results; they are often incompletely recorded, some surgeons decline doubtful cases and will accept only those which promise definite results. The difficulty and disagreements seem to be about the so-called extrinsic type of case. According to reports, there seems to be a controversy between operation and irradiation. With the incomplete reports of irradiation where the radiologist claims beneficial results, the erroneous belief has arisen that almost all, if not all, cases of extrinsic cancer should be irradiated in preference to surgery, and that the radiologist should be consulted as to the advisability of irradiation before surgery is attempted. Such fallacy I have attempted to show in my tables. I feel that surgery still holds a very important place in the battle against extrinsic cancer of the larynx and pharynx. This statement has been confirmed by others who are doing extensive neck surgery.

TABLE 4. EXTRINSIC CANCER OF THE LARYNX.

140 Cases of Extrinsic Cancer of the Larynx and Pharynx Operated Upon or Given Some Form of Irradiation Showing Results by Years.

	Radiation Years						Surgery Years					
Site of Cancer	No.	1	2	3	4	5	No.	1	2	3	4	5
Epiglottis	9	7	1	0	1	0	12	3	1	1	2	5
Epiglottic fold	11	9	2	0	0	0	13	7	3	1	0	2
Arytenoid	7	5	2	0	0	0	6	2	1	1	0	2
Pyriform sinus	25	22	3	0	0	0	20	8	6	1	0	5
	52	43	8	0	1	0	51	20	11	4	2	14
Postcricoid	0	0	0	0	0	0	16	10	4	0	0	2
Lateral wall pharynx	0	0	0	0	0	0	13	6	2	2	1	2

Radiation three years and over, 1. Over five years, 0.

Surgery three years and over, 39 per cent. Over five years, 27 per cent.

TABLE 5. COMBINED RECAPITULATION.
251 MAJOR OPERATIONS NOT INCLUDING 51 TACHEOTOMIES.

OPERATIVE MORTALITY 1.9 PER CENT.

Alive and well under 1 year	27	Died from other causes who	
Alive and well 1 to 5 years	42	Lived under 1 year	20
Alive and well 5 to 10 years	25	Lived from 1 to 5 years	9
Alive and well over 10 years	37	Lived from 5 to 10 years	9
		Lived 10 years and over	15
	131		53

Alive and well	131
Died from other causes	53
Successful cases	184 or 73 per cent cures.

When we consider in general the results by surgery, it is fair to ask ourselves what are the results without surgery. At the present time two conclusions present themselves: first, we have technical methods which seem soundly established and capable of progressive refinement and extension; second, we can say that in cases carefully but not too rigorously

selected as favorable, we can look for satisfactory results as to the disease, cures by drastic but not very dangerous operations which have no serious disability. I cannot agree with the statement that these operations are mutilating. In our follow-up we rehabilitate the laryngectomized patient in the use of the esophageal or pharyngeal voice. It is gratifying to see how very promptly these patients readjust themselves, returning to their former occupations and enjoying life.

The preoperative and postoperative care of these patients is of utmost importance. It is only with the meticulous care and the coordinated efforts of the surgeon, pathologist, anesthetist, assistants and nurses that it is possible to show the results as I have. These operations are not one man's work; they involve teamwork of the highest type, and it is this team's work that shows the low mortality rate.

I believe that radical surgery saves many lives that would have been sacrificed if more conservative forms of surgery had been performed, and I still believe that more lives can be saved by surgery, if diagnosis is made earlier in the epilaryngeal or extrinsic cancer of the laryngopharynx.

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MISSISSIPPI VALLEY MEDICAL SOCIETY MEETS AT ST. LOUIS, SEPT. 28, 29, 30, 1949.

The Fourteenth Annual Meeting of the Mississippi Valley Medical Society will be held at the Jefferson Hotel, St. Louis, Mo., Sept. 28, 29, 30, 1949, under the presidency of Dr. Alphonse McMahon, of St. Louis University. At the recent current meetings of the Society and board of directors, the following officers were elected: Dr. Nathaniel G. Alcock, Iowa City, Iowa, President-elect; Dr. Wendell G. Scott, St. Louis, Mo., First Vice-President; Dr. Charles F. Harmon, Springfield, Ill.; Second Vice-President; Dr. John I. Marker, Davenport, Iowa, Third Vice-President; Dr. Harold Swanberg, Quincy, Ill., Secretary-Treasurer; Dr. Ralph McReynolds, Quincy, Ill., Accounting Officer.

INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY, LONDON, 1949.

The British Association of Otolaryngologists is organizing the Fourth International Congress of Otolaryngology, to be held in London from July 17 to July 23, 1949. There will be further meetings, for those who wish to go, at Oxford, Cambridge and Edinburgh on July 25 and 26. It is hoped that a full academic program will be arranged, and also various social functions.

The secretaries of the National Otolaryngological Societies have been circularized and asked to send a list of their members for individual notification. Should any association not receive this letter, they should communicate with the General Secretary, F. C. W. Capps, F.R.C.S., 45, Lincoln's Inn Fields, London, W.C. 2.

THE ROLE OF THE UPPER RESPIRATORY MUCOUS MEMBRANE IN THE PATHOGENESIS OF POLIOMYELITIS.*

W. LLOYD AYCOCK, M.D. (by invitation),
Boston, Mass.

Clinically, there is an unbroken gradation from frank paralytic poliomyelitis through milder and nonparalytic forms to suspected abortive infections. There are epidemiological space and time relationships between clinically recognized cases which imply intervening milder infections or "healthy" carriers. There is an extensive development of immunity in the general population, as shown by virus neutralization tests; and finally, the virus is actually detected not only in recognized cases but also in milder suspected illnesses and healthy individuals. All these observations afford conclusive evidence to the effect that the virus is widely disseminated and that the paralytic disease is the exceptional manifestation of infection with the virus; furthermore, there are reasons for believing that this exceptional but serious consequence in what is ordinarily an entirely benign and trivial virus infection may be determined not so much by the circumstances of exposure to the virus but by factors which make for susceptibility in a certain few of the many who are exposed.

As long ago as 1910, Sheppard, having in mind a "tonsillectomy campaign" which coincided with an epidemic of poliomyelitis, and citing one case, included tonsillectomy as one of

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a number of possible predisposing circumstances in the causation of poliomyelitis. As is frequently true of diseases of the locomotor system, stresses, strains and traumas were most in favor as contributing causes. Many such circumstances were noted from the most trivial to the most severe accidents, and those ranging in time from the moment of detection of the illness to as far back as the memory extended. Attention was focused most sharply upon circumstances immediately preceding the illness, and indeed many of the falls or injuries noted, rather than being preceding circumstances, were perhaps the result of the already present but unrecognized disease.

It is not surprising, then, that surgical injury of the nasopharyngeal mucous membrane, but one of the many preceding circumstances which were being considered as of possible etiologic interest, did not at this time become the subject of more exhaustive study; nevertheless, Sheppard seemingly intuitively singled out tonsillectomy from the heterogeneous groups of wounds, falls, injuries, exposures, overexertions and so forth when he wrote: "It might be reasonable to suggest at this time that tonsil and adenoid tissue may offer a resistance to microbic invasion. If this is the case, it might seem on *a priori* grounds injudicious to remove an inflamed tonsil, especially in the presence of an acute epidemic infection characterized in many instances by involvement of the upper respiratory passage. . . ." Occasional cases subsequently mentioned in the literature as occurring at suspiciously brief intervals following tonsillectomy have usually been explained by conjectures such as, that the surgeon himself might have been a "carrier" of the virus and infected his patient.

In 1929, Ayer reported "as of possible import in the epidemiology of poliomyelitis" the observation of nine cases of bulbar poliomyelitis which followed tonsillectomy by from five to 10 days. At the same time, Aycock and Luther reported a controlled study which revealed an unmistakable relationship between tonsillectomy and the occurrence of poliomyelitis, in that an excessive number of cases of bulbar poliomyelitis were found to have occurred at a specific time interval following

the operation. Of 714 cases, 217, or 30 per cent, had had a tonsillectomy at some time. By comparison with other estimates for school children of corresponding ages, it did not appear that either presence or absence of tonsils was a predisponent to poliomyelitis; however, a disproportionate number of the poliomyelitis patients who were studied had had the operation within a year of the onset of the disease; and of these, a disproportionate number had had the operation within a month of the onset of the disease. Of the 16 cases in this latter group, all had had the operation between seven and 18 days before the onset of their illness; furthermore, all these cases were of the bulbar type, which, in general, comprises only something like 10 to 15 per cent of reported cases of poliomyelitis. Finally the interval of time between the operation and onset of the disease agreed exactly with the length of the incubation period of the disease.

Thus, the relationships here were such, in the light of epidemiologic knowledge of the disease, particularly that related to the widespread occurrence of subclinical infection (or harmless harborage) of the virus, that they could only be interpreted as indicating that in an individual harboring the virus the surgical procedure sometimes initiated the clinical disease to become manifest in the bulbar form after the incubation period characteristic of the disease.

Since these earlier reports appeared, almost every sizeable series of cases of poliomyelitis includes a small, to be sure, but nonetheless significant proportion of cases of the type under discussion. For example, Eley and Flake in 1938, made a study of recent tonsillectomy in 418 consecutive patients with poliomyelitis admitted to the Infants' and Children's Hospitals of Boston. They found that of the 287 spinal cases, eight, or 2.7 per cent, had had tonsillectomy at some time within 30 days — seven, eight, 10, 24, 25, 26, 29 and 30 days — preceding the onset of the disease. In striking contrast, 17, or 13 per cent, of 131 bulbar cases had had tonsillectomy — all between the tenth and twentieth day preceding onset of the disease; yet, in 1948, opinion varies all the way from the point

of view that there is no relationship between tonsillectomy and poliomyelitis to the belief that all surgical procedures on the upper respiratory passages should be suspended during the "poliomyelitis season."

As a matter of fact, the two diametrically opposed points of view seem to take their origin not so much in any divergent evidence as in convictions stemming, on the one hand, from a willingness to put any precautionary measure against poliomyelitis before other medical considerations; and on the other hand, an unwillingness to let anything interfere with convenience, if we are to believe such often heard statements as "getting ready for school" applied to the operation.

From the nature of the situation, with a very sizeable proportion of the population experiencing a tonsillectomy at one time or other and only a very small fraction of the population ever suffering an attack and an even smaller fraction suffering the bulbar form of poliomyelitis, any actual association between the two would not be strikingly apparent in even a very large series of tonsillectomies, while it might easily be seen in a much smaller series of cases of poliomyelitis. In other words, one could do many tonsillectomies without encountering such a case, but one would not have to see many cases of poliomyelitis before seeing bulbar cases following the operation by a specific interval.

Furthermore, the evidence usually obtained in any statistical survey may not be at once convincing for the reason that the hazard of the disease in recently tonsillectomized individuals is numerically small, even though much larger than in the population in general. In large surveys, such as the one conducted by Dr. Cunning in 1946-1947, the excess in the frequency of recent tonsillectomy in bulbar cases over that in spinal cases and the excess in the frequency of tonsillectomy over a large assortment of other "surgical" procedures when distributed over a period of 60 days amounts to a statistical dilution which makes it not at all impressive. Had these studies gone one step further to test the crucial question whether

tonsillectomy preceding bulbar cases was significantly concentrated at around 14 days preceding the disease, the question of a relationship would have been settled.

Thus, the question of the relationship and what measures to take becomes an annual debate, generally between otolaryngologists and poliologists — not so much because of any fundamental disagreement between them, but because they are looking at the same problem from such widely divergent angles. At the same time, there is a tendency toward continued study by broad population survey methods which, by their very nature, are not likely to afford an answer to the question which would be any more decisive (because any differences shown by them are likely to be fractional) than are the few detailed studies which are already available—or better still, than the clinical experience seen in any sizeable number of cases of poliomyelitis, as for example, the group studies by Eley and Flake.

On the other hand, surveys of the question limited to periods of time and to places where poliomyelitis is prevalent do bring to the surface a numerical risk which is largely hidden in the more general survey figures. Such information is of value in affording an actual numerical basis for any "policy" with reference to the institution of precautionary measures.

One such survey is that of Anderson. In 1943, an epidemic of poliomyelitis occurred in Utah, in which the attack rate exceeded that of any other state that year. As a result of certain local circumstances, practically all cases (136 out of a total of 400) reported by physicians as bulbar or respiratory were admitted to one hospital. About one-third of these were of the bulbar or bulbospinal type. The frequent history of recent tonsillectomy in these bulbar cases led to a study of the relationship in the ascending part of the whole epidemic in the state. During the three-month period (July, August, September), 4,199 tonsillectomies were performed in the child population under 16. Seventeen of these individuals developed poliomyelitis of the bulbar or bulbospinal type within 30 days following the operation. During the same period, 232

cases of poliomyelitis occurred in the remaining child population (156,000), but only 39 of these were of the bulbar type. The incidence of the disease in the recently tonsillectomized was over two and one-half times as high as in others; and the chance of the bulbar type in the recently tonsillectomized was 16 times as high as in others.

Thus, this evidently complete and accurately done survey reveals a hazard of bulbar poliomyelitis following tonsillectomy when done in an intense epidemic situation of such magnitude that it calls for the institution of any feasible precautionary measure which may be available. At the same time, this hazard, impressive as it is when expressed in terms of poliomyelitis, from the otolaryngologists point of view might not appear on the surface to be alarming.

As shown by the Utah survey, somewhat less than one in 200 tonsillectomies was followed by bulbar poliomyelitis. When we remember that the figures here are based on an unusual epidemic situation, this ratio is undoubtedly of the highest order. As epidemics of poliomyelitis go, it would certanly be much lower on the average.

The intensity of debate in the case of this newly learned surgical hazard has seemed to far exceed the concern over numerous other hazards well known to accompany various medical procedures. Many of these may be fully as important as the hazard of poliomyelitis following tonsillectomy, both as to frequency and severity. Sound practice, recognizing a hazard which may be as high as one in 200 (or perhaps we might say even as low as one in 200), sets about to eliminate, avoid, minimize or even accept the risk, depending in the particular situation upon the relative importance or urgency of the procedure itself and the frequency and seriousness of its accompanying hazard; but even here, the surgeon is not concerned with statistical frequency alone. The timing of the operation, his choice of anesthetic, and many other preparatory or precautionary procedures are determined by circumstances in the individual situation, and by the desire to render the best medical service to the individual patient.

All this is just another way of saying that the risk of bulbar poliomyelitis following tonsillectomy *most of the time in most places* is almost nil; but that *for short periods of time in a few places* (epidemics of poliomyelitis) it is a risk to be avoided if it can be done without inviting some more serious danger to health, and whether or not it interferes with the convenience of the patient, the family, the hospital or the doctor.

Therein lies a solution of the whole controversy which not only would serve what the poliologist and doubtless the public (and even the courts) would consider the primary purpose — the prevention of smaller numbers of cases of poliomyelitis — but would not cause a serious interference with what the otolaryngologist may consider an equally or even more important purpose — the bestowing of the benefits of tonsillectomy upon *much* larger numbers of individuals.

It all comes down to this: in most places most of the time nothing need be done; but in a few places, and for short periods of time, postponing the operation would prevent the occurrence of numbers of cases of an extremely distressing and highly fatal form of poliomyelitis. This in turn leads at once to the question, "In what places and at what times should the operation be avoided?" In the past there has been considerable confusion when in different localities, or even in the same locality, various policies have been adopted by different hospitals or physicians. There are no reasons for supposing that the risk under discussion is in any way dependent upon circumstances within the hospital or would differ with different physicians. On the contrary, the risk is determined by the prevalence of the virus in the community, as evidenced by the occurrence of the disease, and, therefore, would be the same with any hospital or physician contemplating the operation on patients in that community.

In order to avoid the confusion of conflicting policies in the same community situation, and perhaps somewhat as a safeguard against any extreme attitudes in either direction, the nose and throat services in a number of the Boston hospitals are making a practice of following a uniform procedure agreed

upon by them and based on information and recommendations furnished by the State Health Department upon any increase in prevalence of the disease in any of the various areas served by the hospitals.

The adoption of one other more general practice in respect to the timing of the operation suggests itself. From a comparison of the seasonal frequency of poliomyelitis in general and bulbar poliomyelitis following tonsillectomy, it can be inferred that there is a sort of "seasonal epidemic" in tonsillectomy which happens to coincide closely with that of poliomyelitis. This, in a sense, tends to double the risk at the particular season and suggests that this favorite time for the operation, unless there be some distinct contraindication, be changed, for example, from late summer "before school opens" to early summer "after school closes," but before poliomyelitis begins.

Another aspect of the tonsil-poliomyelitis relationship, one which has received far less attention, should be mentioned, in that it shows the very definite influence of the tonsil on an infectious process. Without any relationship to the tonsillectomy operation itself, the data in all of the surveys which have covered this point show that the absence of tonsils favors the occurrence of the bulbar rather than the spinal form of the disease. This does not mean, however, that the absence of tonsils necessarily predisposes one to the disease. The survey by Top and Vaughan, in Detroit, suggests that the incidence of poliomyelitis may be higher in tonsillectomized individuals, but most of the surveys do not lend themselves to analysis of this question. It is clear that the presence or absence of tonsils has a definite influence on the type of the disease which is to occur. Perhaps this is more strikingly shown in an analysis of multiple cases in families where cases of both types occurred at the same time. In such a situation many epidemiologic factors can be assumed to be the same. The cases are in all probability the result of exposure to a single strain of virus. Environmental circumstances are, in general, the same and can be excluded as determining factors. In 46 fami-

lies where two or more cases occurred, it was found that of 25 bulbar cases tonsillectomy had been performed recently or previously in 17, while in 76 spinal cases only 10 had been tonsillectomized at any time previously. It is not meant to imply that the data in these surveys in any sense comprise any argument against tonsillectomy in general, but it is presented as a clear indication that the tonsil has a definite influence on the infectious process.

Perhaps it should be emphazised that the relationship between tonsillectomy and poliomyelitis plays a relatively small part in the epidemiology of poliomyelitis. In other words, complete compliance with any precautionary measure which may be indicated by these studies could result in the prevention of only a small proportion of cases of the disease. I think of more far-reaching importance in the study of the disease is the fact that the occurrence of the bulbar type of the disease after tonsillectomy and with greater frequency in tonsillectomized persons affords the most striking illustration which is available of the epidemiologic principle that the frank paralytic disease in the few of the many who are exposed to the virus may be determined more by circumstances in the host than in the circumstances of exposure to the virus.

It is believed that a closer study should be made in the field of otolaryngology of the as yet unexplained mechanism through which the tonsil relationships play a part in susceptibility to poliomyelitis. A clearer understanding of the manner in which the tonsil influences susceptibility in a small fraction of cases might well give some indication of the nature of other factors which play a part in susceptibility.

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TWO CASES OF ACUTE DIFFUSE LABYRINTHITIS FOLLOWING ACUTE OTITIS MEDIA.*†

HARRY ROSENWASSER, M.D.,
New York, N. Y.

Fifteen years ago, Dr. Friesner and I[1] published a paper entitled, "Treatment of Labyrinthitis," in which we presented the historical background, reviewed the literature and set forth our viewpoint in the treatment of all forms of labyrinthine infections. At that time, we stated that, despite the voluminous literature with its innumerable reports of cases, wide differences of opinion existed regarding surgical therapy in some forms of labyrinthine disease. We reserved for the last the consideration of that form of labyrinthitis concerning which the widest differences of opinion existed, namely: diffuse labyrinthitis occurring during the course of an acute otitis media.

In that exciting era prior to chemotherapy and penicillin therapy, Voss,[2] Scheibe,[3] Alexander,[4] Phillips,[5] Page,[6] Milligan,[7] Smith,[8] Perkins,[9] Cunning,[10] Ramadier[11] and many others advocated conservatism in the care of labyrinthitis occurring with or shortly after acute otitis media. Scheibe even urged immobilizing the head and body with starch bandages. On the other hand, an equally large group of competent observers, including Hinsberg,[12] Neuman,[13] Bárány,[14] Leidler[15] and others, advocated an extremely radical course. In discussing the indications for labyrinthectomy in cases of acute laby-

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†From the Otolaryngological Service of Dr. R. Kramer, the Mt. Sinai Hospital, New York, N. Y.

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rinth destruction occurring during the course of an acute otitis media, Lund[16] stated that there was no doubt that satisfactory results were obtained neither by the radical surgical therapy nor by conservative measures.

If one draws any conclusions at all from these divergent views, it must appear that cases of acute diffuse labyrinthitis presenting primarily identical symptomatology differ in the widest possible degree in their tendency to become limited and localized and to heal, and not to extend to the intracranial contents. If this is true, and I do believe it is, it follows that each case of acute diffuse labyrinthitis must be considered individually as to therapy. If this were not true, these differences of opinion on the part of competent observers would be utterly unintelligible.

Acute diffuse manifest labyrinthitis has been customarily divided into two groups: *1.* serous and *2.* suppurative, a grouping which has resulted from an attempt to classify clinically identical phenomena on the basis of pathologic evidence. Otologists often have fallen far short of the actual differentiation between these two states because the clinical criteria advanced have been inadequate to establish accurately such differentiation. The inability to differentiate between serous and suppurative labyrinthitis is not necessarily serious, providing that as a result of careful clinical observation and experience, one substitutes for it knowledge as to which form of labyrinthitis is grave.

In this communication, I propose to discuss briefly the acute diffuse labyrinthitis which occurs with or shortly after acute otitis media and to present in some detail a report of two recently treated cases. Some of our previously held concepts in the handling of this serious otitic complication may require re-evaluation, in the light of the known advantageous effect resulting from the early use of chemotherapy and penicillin therapy in adequate dosage.

Case 1 (No. 560555): An adult male, aged 26 years, was admitted to the Mt. Sinai Hospital, Feb. 14, 1947, acutely ill, with temperature 105°, headache, dizziness and vomiting. In 1936, he was found to have moderately advanced tuberculosis. One year later, he was again hospitalized

at the Medical Center, New York, where, after careful and thorough study, the diagnosis of periarteritis nodosa was made. In 1938, and 1940, he was again readmitted and the diagnosis was confirmed clinically and by biopsy. Except for occasional mild upper respiratory infections, the patient remained in good health until three days before his present admission, when he caught "cold."

Because of his past medical history, and because he had been advised to care for even the most insignificant infection, the patient remained at home. The next morning he had pain in the left ear, which persisted throughout the day and in the evening, there was a spontaneous sanguino-purulent discharge from the left middle ear. He was promptly placed on sulfadiazine therapy by his family physician. The patient was quite sick, the temperature was 103° and there was restlessness, irritability and nausea. Forty-eight hours after the onset of the acute otitis, the patient was given penicillin in addition to his sulfadiazine medication and at this time I was asked to see him.

Examination revealed an acutely ill male, nauseated, vomiting and complaining of severe generalized headache. Motion of the head aggravated the symptoms. The patient was easily aroused and quite cooperative. An inexhaustible spontaneous nystagmus directed horizontally to the right was noted. With the Bárány noise apparatus, in the right ear, there was *no hearing demonstrable in the left ear.* There was some stiffness of the neck, but no other frank meningeal signs. A few asthmatic wheezes in both sides of the chest and definite evidence of an acute pansinusitis were found.

The left external auditory canal was filled with pus which reappeared promptly after wiping it away. There was no sagging of the bony canal wall, no mastoid tenderness and all the normal drum landmarks were absent. The right drum was slightly full posteriorly; the landmarks, though indistinct, were discernible.

Laboratory Findings: Hemoglobin content, 12.4 gms.; white blood cell count, 15,850, with 86 per cent polymorphonuclear cells and 14 per cent lymphocytes. Serology and repeated urinalysis were negative. Culture of the discharge was reported staphylococcus albus B. Blood pressure determinations throughout the patient's stay in the hospital revealed a hypertension fluctuating between 194/120 to 160/112. Roentgenograms showed extensive pneumatization of both sides. On the right side there was diffuse haziness, particularly in the periantral region and over the knee of the sinus. The right lateral sinus was more prominent than the lateral sinus on the left. There was no definite evidence of destruction.

Course: The patient was put on what was regarded by some as unnecessarily large doses of crystalline penicillin, 500,000 units intramuscularly q. 3 h., and also given sulfadiazine (1 gr. per pound) intravenously. The penicillin therapy was maintained for two weeks, at the end of which time the patient still had some objective vertigo but no longer had any nystagmus. At this time unmistakable return of hearing was apparent in the previously completely deafened right ear. The patient was eating and sleeping well and no longer had headaches. On March 3, 1947, there was noted further progressive improvement in hearing. On March 9, 23 days after admission, all medication was discontinued, improvement continued, and on the thirtieth day after admission the patient was discharged.

Follow-Up: This patient has remained well, the hearing is now excellent; has normal cochlear and vestibular responses.

Comment: I believe we are dealing with an acute suppurative otitis media with extension of the inflammatory process to the labyrinth via the oval or round window. In the absence of any clinical evidence of mastoiditis and in the absence of convincing evidence of meningeal involvement, there seemed to me to be little justification for surgery. The indication for the use of massive doses of penicillin and sulfadiazine appeared logical in view of the potential serious intracranial complications. Since one cannot definitely distinguish between a diffuse serous or suppurative labyrnthitis on the basis of the functional tests, the assumption must be made, in retrospect, that we were dealing with a diffuse serous labyrinthitis because of the return of function. Whether the early employment of massive doses of sulfadiazine and penicillin can overwhelm and sterilize an early suppurative invasion of the labyrinth and result in a cure, with return of function, is conjectural and intriguing. The reports of the experience of others may throw some light on this possibility.

Case 2 (No. 585393): Patient, F. V., aged 10 years, was admitted Aug. 7, 1948, to the Ear Service (Mt. Sinai Hospital, New York) with a history of a discharging ear and high fever of three days' duration. Of significance in the past history was the occurrence of mumps at three years of age and 20 per cent loss of hearing in the right ear (nerve type) noted when tested audiometrically in the public school system. The patient was swimming one week before admission. He was perfectly well until three days prior to admission, when he complained of pain in the right ear and had a temperature of 103°. The following day there was a scant spontaneous discharge from the right ear, some slight nausea but no vomiting. The patient was brought to the emergency room, given sulfadiazine therapy and sent home, with instructions to return for further observation.

The following day when seen he was quite sick and was promptly admitted to the Ear Service. He was drowsy, complained of severe frontal headache and vomited persistently. While in the ward, in a few hours, he became rapidly worse.

Examination that evening revealed a very sick child; temperature, 105°; pulse, 152; respirations, 40 per minute. Oculi fundi were negative, pharynx injected, tonsils enlarged and inflamed, the heart and lungs were negative.

The left drum was normal; the right drum was inflamed throughout; the short process was distinctly visible. There was no mastoid tenderness, no change in the bony canal wall. There was a spontaneous nystagmus directed horizontally to the left, inexhaustible and present even on contral gaze. *There was no hearing in the right ear, using a Barany noise apparatus in the uninvolved left ear.*

There was marked nuchal rigidity, the child was drowsy and somewhat irrational. Lumbar puncture, which was promptly performed, yielded cloudy fluid under increased pressure, and contained 6,000 cells, mainly polymorphonuclear leucocytes, with Gram positive organisms on smear. Crystalline penicillin, 10,000 units, was instilled intrathecally before the cell count and smear were reported, and without awaiting the report of the penicillin sensitivity test.

Laboratory Data: Culture of the spinal fluid revealed a pneumococcus type XII. The pneumococcus type XII was found to be susceptible to 0.02 units of penicillin (lx's as resistant as standard). Blood culture, blood Wassermann reaction and repeated urinalysis, Roentgenograms of the chest and mastoid were entirely negative. Hemoglobin determination revealed 12.5 gm.; white blood count, 23,650, with 80 per cent staph cells; 9 per cent segmented polymorphonuclear leucocytes; 7 per cent lymphocytes and 3 per cent monocytes.

Course: The patient was given large doses of sulfadiazine intravenously and 50,000 units of crystalline pencillin intramuscularly every three hours. After 24 hours, the temperature subsided to 102°, there was abatement of the marked hyperexcitability; the photophobia, however, persisted. The Kernig and Brudzinsky signs were still present. At this stage it was obviously vitally important to ascertain whether there was vestibular function present. Caloric test was performed, using hot 1:5,000 bichloride of mercury solution. There was a prompt response noted for nystagmus to the involved side and influencing the pre-existing spontaneous nystagmus directed horizontally to the left.

It was, therefore, assumed that we were dealing with a labyrinth that was still functioning. It was further assumed at that time that there may have been a dichotomous spread of the infection *1.* from the middle ear to the meninges, either through vascular channels or dehiscences, and *2.* from the middle ear via the oval or round window to the labyrinth. This type of dichotomous extension has been heretofore described by Dean and Wolfe[17] and others. It seemed unlikely that a meningitis with 6,000 cells, mainly polymorphonuclear leucocytes, with pneumococci on smear and culture, was apt to result as a direct extension from a labyrinth which still retained caloric irritability.

Since there were neither clinical nor Roentgenographic signs of mastoiditis, and since there was some evidence which seemed to point to a diffuse serous rather than a diffuse suppurative labyrinthitis, it was felt that there was no indication to surgically attack the labyrinth or mastoid bone. Intensive medication consisting of sulfadiazine and penicillin was continued for three weeks. The meningeal signs rapidly subsided. A second lumbar puncture at the end of one week of therapy revealed 30 lymphocytes and no organisms were noted on smear or culture. Because of the marked improvement clinically and in the spinal fluid findings, further intrathecal use of penicillin was not deemed necessary. At this time, the patient was symptom-free, there was only an occasional mastoid jerk visible on extreme lateral gaze. The sulfadiazine was discontinued and the dosage of penicillin was gradually lessened and finally stopped. Five weeks after admission, the patient was up and about, asymptomatic, eating sleeping well; however, it was evident that there was *no hearing* present in the right ear and repeated caloric tests revealed a calorically unirritable labyrinth.

Comment: Whether the fact that the offending organism was a type XII pneumococus played any rôle in the resultant total loss of function, I do not know. It is important to identify the infecting organism promptly in every instance to determine its relative sensitivity to penicillin that adequate dosage may be estimated and given. Complications following recovery from pneumococcal meningitis have not been adequately described according to Waring and Weinstein,[18] who state that permanent neurologic damage in young children is common and often severe. In 433 cases, Waring and Weinstein found the pneumococcus type XII in six cases. Another point of interest stressed by these observers was that relapse was an event which occurred quite regularly with the sulfonamide treatment of meningitis, due to pneumococcus, and it occurred soon after therapy was discontinued or dosage diminished. This patient at one stage of his infection had a total loss of hearing but still maintained caloric irritability. Despite cure of his bacterial meningitis, the vestibular function ultimately was completely lost.

Grove and Strauss,[19] in 1941, reported a case of diffuse labyrinthitis and meningitis following acute middle ear suppuration, treated by radical mastoidectomy and massive dosage of sulfonamides. The patient recovered, with a partly functioning labyrinth. Unterberger,[20] in 1941, reported a similar case, which, according to the prechemotherapy-day attitude towards treatment, would likely have been subjected to labyrinthectomy. The patient, however, was treated with massive doses of chemotherapy and recovered with partial restoration of labyrinthine function. Case 1 herein reported is an instance of diffuse labyrinthitis, in a patient with known healed periarteritis nodosa, who recovered completely with normal vestibular and cochlear function.

Siirala[21], from Helsinki, in 1946, reported four cases of labyrinthogenic meningitis treated with sulfonamide therapy. In three of the four cases reported, the onset was initiated by an acute otitis media. In each of these three cases, the meningitis appeared simultaneously with the symptoms of involve-

ment of the labyrinth, which was involved totally. All four cases reported and treated by Siirala ultimately eventuated in dead labyrinths. I believe it worth noting that in none of these cases was sulfonamide therapy started at the very onset of the acute infection in adequately large doses, nor do I believe that Case 2, reported by me, received adequate therapy early enough. Sulfadiazine and penicillin therapy in this type of case, to be successful and hold some hope for complete recovery with a functioning labyrinth, must be given very early and in very large doses. Since there are so few cases, and since the experiences of others is not readily available for study in numbers sufficient to draw conclusions, I have arbitrarily given one-half million units of crystalline penicillin intramuscularly every three hours with the full realization that this might be far in excess of what might ultimately prove to be necessary.

It is perhaps unfortunate that we have become accustomed to figuring dosages of penicillin in thousands and hundreds of thousands of units. There would, I am sure, be considerably less reluctance to the employment of penincillin in cubic centimeter doses than there often seems to be when doses of one-half million units are suggested. Some of the factors that must be considered in promptly deciding dosage, in my opinion, in order of their importance are: *1.* the duration of the labyrinthine infection; *2.* the identity of the offending organism; *3.* the susceptibility of the infecting organism to the antibiotic employed; *4.* the accessibility and extent of the infection; and *5.* whether there is meningitis. Initial doses of one-half million units are suggested while awaiting laboratory data.

Whether the treatment of labyrinthitis and meningitis following an acute otitis media results in a cure of the meningitis, as in Case 2, and leaves the patient with a nonfunctioning labyrinth, one must speculate whether earlier and more intensive treatment might have resulted in cure of the meningitis with a viable labyrinth. The healing or reparative processes in a labyrinth frequently results in a loss of func-

tion (total or partial) in contrast to the healing of infections elsewhere in the body, *i.e.*, peritoneum, orbital cellulitis, etc., where functional impairment is negligible or unimportant. It is, therefore, particularly important in the care of acute labyrinthitis following acute otitis media that the optimal dosage of penicillin and sulfadiazine be quickly determined and given. Time is most important at the onset of the disease, and I suspect largely determines ultimate residual function. Finally, in the cases which recover, but have *no* labyrinthine function, one must consider the possibility of a latent suppurative focus having been established in the labyrinth with its inherent danger of late intracranial complication. It behooves one to observe such patients for long intervals before this potential menace can be discounted.

Conclusions: In this era, with its sharply curtailed incidence of suppurative disease of the mastoid and temporal bone, otologists still occasionally are confronted with cases of acute labyrinthitis and meningitis, or acute labyrinthitis with meningeal irritation occurring early after or together with the onset of an acute otitis media.

In the prechemotherapy days, the prognosis in these cases, whether they were treated by conservative or radical surgical measures, was not very encouraging. With the addition of sulfadiazine and penicillin to our armamentarium or therapy, the outlook for preservation of life is no longer the grave concern of former years. I submit that the objective of the otologist should now be preservation of life and preservation of function as well, and this objective seems possible of attainment by the early use of sulfadiazine and penicillin in adequate dosage.

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VESTIBULAR EXPERIENCES.*

ISAAC H. JONES, M.D.,
Los Angeles, Calif.

To give experiences one must, unfortunately, be entirely personal. The experience of another would require another article—by him.

With me, in studying the internal ear vestibular studies are routine, especially the turning tests.

These comments will be limited to *1.* office routine; *2.* neuroma of the VIII Nerve; and *3.* vestibular lesion in the brainstem, from streptomycin poisoning.

Recently, two medicolegal cases were studied at the same time. Each had been studied by leading otolaryngologists whom we know and respect — but without vestibular tests. In each, vestibular studies completely reversed the diagnosis. One man, very deaf, had been exposed to loud noise for a long period. The opinions of two otolaryngologists in New York and three in Los Angeles were unanimous — that his cochleae had been damaged by the noise. The first thing to do, of course, was to turn him. After turning there was no nystagmus whatever — not even a quiver of the eyes; so, in less than a minute, it was clear that he suffered from syphilis or some other constitutional disease. Noise, no matter how loud, obviously could not destroy vestibular function. After turning in either direction there was no nystagmus and no sensation of motion; and the caloric tests — douching each ear for

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four minutes with water, temperature 68° — showed a total absence of any responses whatever; there was no nystagmus, motion-sensing, past-pointing, falling or constitutional response. It was also clear that this defect had been coming on for a very long time, because he had never had vertigo or any other vestibular symptom. On receipt of this report the legal decision was reversed and the claim disallowed.

Experiences like this make me realize that, without the vestibular tests, I would come to exactly the same wrong conclusions as my otherwise more intelligent colleagues.

The other man had been examined by four otolaryngologists and the only reason I had the privilege of seeing him was that each of the reports showed such different degrees of deafness that the request was "to try to determine how deaf he is." He wasn't deaf at all. The hearing was good in each ear. The reason for his apparent deafness and also for the variability shown in his many hearing tests was that his *attention* varied. The injury produced psychic trauma — not acoustic trauma. When the caloric tests were made the following occurred: While the right ear was being douched (completely masking out hearing from this ear) he responded to all questions spoken in a soft voice. When he began to feel "dizzy" we said in a quiet and kindly voice, "Does the water make your ear feel too cool? Do you feel that things are turning around?" His perfectly natural responses demonstrated that he had excellent hearing in this "deaf" ear. Similarly, while the left ear was being douched he responded to questions promptly and accurately. Above my desk hangs a wood-duck. While my back was turned to the patient he asked, "Isn't that a wood-duck?" Without turning my head I said softly, "Yes, what do you know about wood-ducks?" Deeply interested, he related that on one occasion he had shot down 11 and that he was much upset when he found out the law did not permit more than one wood-duck to be killed by any one person in one year. Again we realized that his hearing was very good indeed; but later, while he was sitting in a corner for the obvious purpose of having his hearing tested, I approached him from a 20-foot distance. He did not answer

questions when my voice was loud even at a distance of four feet. Obviously his hearing trouble was due to inattention. He could not concentrate on the matter at hand. Evidently the blast of air to which he had been subjected had touched off this set of mental reactions. There was no thought of malingering. The man showed every willingness to cooperate to the fullest extent. He repeatedly said, "All I know is that I was able to do good work before this thing happened and now I am no longer able to do such work." He was not only sincere but naive. In this case we were put on the right track because the peculiar sensations from the turning, and especially from the douching, took his mind off his troubles — then he heard well. Our suggestion to the court was to have the proper person explain to him the real nature of his trouble. We felt that if this could be done he would not only have satisfactory hearing but should come to realize that he does have good hearing.

We vary as to our ideas of what is essential—which is natural. I, for one, would not know what to do without the routine vestibular tests in any problem that concerns the internal ear. If the ophthalmologist used the ophthalmoscope and took the fields of vision—and yet studied only one-half of the retina—we would not only be critical of his technic but would realize that he might be missing the main lesion. If I were considering having a fenestration operation on myself, I would want the fullest information as to the precise condition of my internal ear. Why limit the study to the cochlea? Why not find out all we can, instead of being satisfied with "just enough"? One might say, "I have never had a turning chair and yet I get along all right." I couldn't. Some feel that the caloric test is all-sufficient. Not for me. Often the turning shows responses very different from the caloric; we do not know why, although we suspect it is because turning produces a more intense and less prolonged stimulus.

All of us have babies brought to us who appear to be deaf. We turn the mother with the child in her lap. The mother holds the child with the head forward toward her knees, with her hands over the baby's eyes. At the end of the turning the

mother keeps her eyes shut, but lifts the child. If there is no tendency for the child to fall the evidence is strong that both internal ears are markedly involved.

In court we have frequently demonstrated that a lesion of the internal ear was recent. When the facts are explained to the jury, they are always accepted at face value. Recently a woman showed a lesion of the left internal ear. We explained that it was recent — because turning to the right showed almost no responses, whereas turning to the left showed excellent responses. We explained that after a few months, although the lesion might remain the same, the responses would have become equalized. She would then show the same response after turning in either direction.

What do we mean by vestibular tests? We certainly do not mean some of the tests that are now used, such as using a certain amount of water or just one squirt of ice water. Surely such tests are trivial. The neurologist does not limit himself to the cremasteric reflex and then say, "I have made a complete neurologic examination." For 34 years we have used the same turning and caloric tests. Although the newer tests are more popular and seem an easy way out, yet there are no short cuts to good work and a complete study. Our technique has always been as follows: *Spontaneous:* Nystagmus and pointing. *Turning:* The duration and amplitude of nystagmus; the number of seconds of motion-sensing; constitutional responses. *Caloric:* Water, 68°; how long it takes the douching to produce the nystagmus; pointing tests; head back (testing horizontal canal), note nystagmus and take past-pointing; falling test; then head back again, noting duration of nystagmus; note constitutional responses.

TUMOR OF THE VIII NERVE.

It is not generally known that one-tenth of all brain tumors grow on the VIII Nerve. If a patient is very deaf in one ear, the first thought should be of a possible tumor. In some the first symptoms are auditory — hearing defect and tinnitus; in others, although less often, the first symptoms are vestibular.

The phenomenon-complex which we brought out in 1915 has been confirmed by our experiences since. *1.* On the side of the tumor a loss of auditory and vestibular function. *2.* On the opposite side normal auditory function, normal responses from the horizontal canal, but absence of responses from the vertical canals. The usual sequence: During the early period of the development of the tumor a gradual or sudden loss of auditory and vestibular functions. In the course of weeks or months one or more of the following findings may appear: *1.* The impairment and later loss of responses from the vertical canals of the opposite side. *2.* Spontaneous vertical nystagmus upwards may be observed. *3.* In some cases the horizontal canal of the opposite side produces normal nystagmus, but later produces a perverted nystagmus.

VESTIBULAR DAMAGE FROM STREPTOMYCIN.

A more recent experience concerns streptomycin poisoning. When this remarkable new drug appeared it was quickly evident that even this rose had its thorn. Its effect on tuberculous and other lesions seemed almost miraculous; but soon many patients were stricken with disturbed equilibrium.

From what one reads, sees and hears, the following symptoms stand out. There are reports of tinnitus; and occasionally of hearing defects, although in most cases some doubt exists because of associated meningitis, syphilis or acoustic trauma from war experience. Certainly there is little or no evidence of the well-known picture of end-organ lesion — vertigo, nystagmus, pallor, sweat, nausea and vomiting. On the contrary these patients complain of symptoms mainly referable to the brain-stem rather than to the periphery. Vertigo, by which is meant "a sensation of turning not in accord with fact," is almost never noted. The patient notes "a queer feeling," which is one of instability or insecurity, when the head is moved. Most of the patients lying quietly in bed and not moving the head have no sensation of movement; it is only on changing position, such as in attempting to get out of bed, that they feel uncertain and go too far in the attempted movement. They tend to fall, but in no special

direction. They "over-fall" in the direction in which they start to move. As one expressed it, "The brakes don't work." If the patient is reading with head quiet on the pillow, the print does not seem to move; it is only when the head is raised that the print appears blurred. On attempting to walk, these patients stagger. They are unable to walk in a dim light or on an inclined surface and tend to fall on turning quickly. Often they do not fall, but fear they will. Later, while riding in an automobile over rough ground, the patient notices that the scenery seems unsteady; but this ceases when the road becomes smooth.

Two years ago the Veterans Administration was startled by the large number that developed ataxia and yet maintained good hearing. I was asked by Dr. John S. Chase, "Where's the lesion?" Without hesitation, I reported, "In the Central Vestibular Tract in the brain-stem." Why such a positive statement? Because of one classic experience.

Dr. William G. Spiller, than who there has never been a greater neuro-anatomist, has given the last word on this subject—in a report by Jones and Spiller* before the American Neurological Society. As these findings are not available in otologic literature they are here included in condensed form. This demonstration of the Central Vestibular Tract, first made evident by this study, was made possible by a sequence of events that may never happen again.

The case of encephalitis presented the following features: An absence of vestibular function, with no impairment of auditory function. All examinations and tests were made just before death, which occurred only 11 days later. The autopsy revealed nothing abnormal — indicating that there were no pressure phenomena and that the lesion was microscopic. Dr. Spiller made over 1,000 serial sections of the brain-stem and for the first time revealed the Central Vestibular pathways.

Before this Central Vestibular Tract was discovered, the auditory fibers had been demonstrated in the brain-stem, and

*Isaac H. Jones, M.D., and Wm. G. Spiller, M.D.: "The Central Tracts of the Nervus Vestibularis." Brain, Sept., 1925.

also the two sets of vestibular nuclei (Deiters, von Bechterew and Triangularis), which receive the fibers from the vestibular portion of each VIII Nerve. There had been no definite evidence, however, to show where the fibers continued from these nuclei in the medulla.

In attempting to discover these pathways, those who have undertaken work on animals have encountered two main difficulties: *1.* Secondary degeneration is interrupted at the next synapse. *2.* There is the obvious difficulty in producing minute lesions in certain portions of the brain-stem without damaging other structures, both distant and adjacent. Throughout these various segments it would be necessary to produce lesions which would involve each neuronal segment in turn. These difficulties appear almost insuperable.

On the contrary, certain pathologic processes occasionally result in small discrete lesions within the central nervous system, which cannot be duplicated by operative work on animals. Such lesions may be produced by thrombosis, multiple sclerosis, or small foci of encephalitis. The proven case reported by Jones and Spiller was of this nature, presenting minute lesions due to encephalitis.

Clinically this boy, age 15, presented a strange picture. He had always been full of energy and a leader in school athletics — particularly in gymnastic work. He could walk on his hands almost as well as on his feet and excelled in basketball, baseball, football and tumbling. He had a good record in his scholastic studies and was in his second year at high school.

Following an acute attack of influenza he complained of fatigue and of being "short of wind." On getting up from a sitting position he was unable to move about without holding on to something to steady himself. The first symptoms noted were incoordination, disturbance of equilibrium and gasping for breath. He had some difficulty in swallowing; no pain. *Neurologic examination by Dr. Samuel D. Ingham:* Patient was well developed and fairly well nourished. Taste and smell normal. Romberg negative. Attempts to cough were ineffectual. Impaired upward movements of the eyes and almost

no convergence. Pupils equal and reacted to light. No loss of voluntary movements of the muscles of mastication or of the face, tongue or palate; no wasting or tremors. Tendon reflexes present in all extremities, slightly excessive but equal on both sides. *Mental condition:* Bright and alert and of more than average intelligence; in complete possession of all his mental faculties. No emotional or psychoneurotic symptoms.

Eye examination by Dr. Theodore C. Lyster: Vision good for distance; near vision, accommodative power and convergence were poor. Eye movements slightly limited in all directions, especially upward, when the eyes began to sag downward and at the same time showed an undulating movement up and down. Disc margins fairly well defined. Maculae clear; blind spots normal. Under homatropine dilatation there was found an error of hypermetropic astigmatism; vision improved to full normal in each eye when corrected.

Ear, nose and throat examination by Dr. Eugene R. Lewis and Dr. Isaac H. Jones: Chronic purulent tonsillitis; chronic pharyngitis; cervical adenitis in the anterior chain — each side; otherwise negative.

Prof. Vern O. Knudsen and Dr. Jones found the audiograms to be normal, including the upper limits. Tests with the amplified voice and amplified whisper showed perfect hearing. No diplacusis or tonal islands. No tinnitus had ever been noted. It was evident that both cochleae and the auditory neural pathways had perfect function.

Vestibular report (see Fig. 1): On looking straight ahead, a spontaneous nystagmus of indefinite character; the eyes were constantly moving but the amplitude was small. The lower and upper eyelids also moved synchronously with the movement of the eyeballs. On looking upward, a vertical nystagmus — but the quick movement was not definite or rhythmic. After turning to right, no nystagmus — other than the already existing spontaneous nystagmus. No past-pointing. Turning to the left similarly failed to produce any nystagmus or past-pointing. Turning to the right with the head forward caused the patient to lean slightly to the right when he sat

up; while turning to the left with the head forward caused him to lean slightly to the left. *Caloric tests:* Irrigation of the right ear, water 68°, for four minutes. No response in nystagmus or past-pointing. Upon tilting his head backward

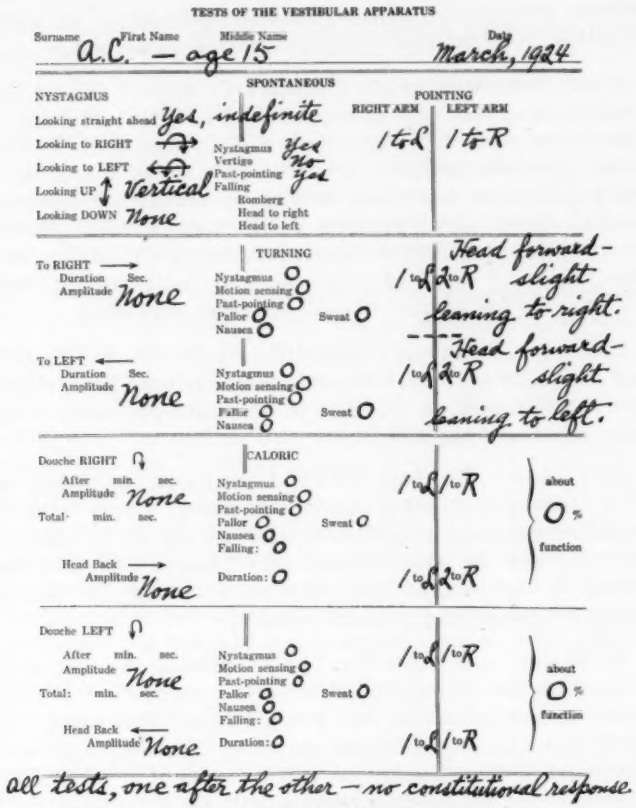

Fig. 1. Encephalitis, localized.

(testing the horizontal canal) there was also no nystagmus or past-pointing. No falling response. On douching the left ear the findings were identical — *i.e.*, no vestibular responses. All the turning and caloric tests were conducted one after the

other and yet there was an utter absence of pallor, sweat or nausea.

There was, therefore, a complete vestibular block, the loca-

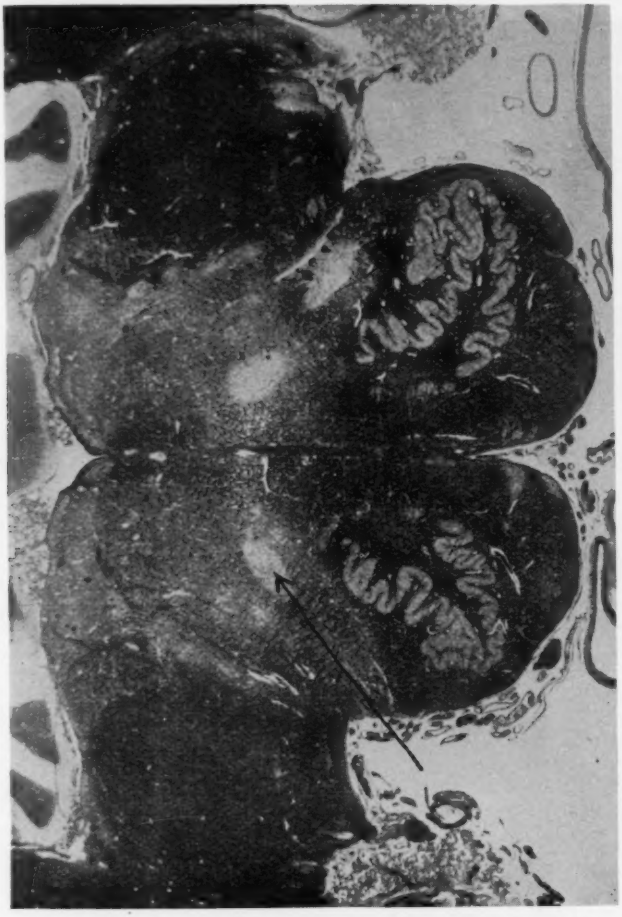

Fig. 2. (Section 9). Separate areas of degeneration on each side of the raphé. (From Jones and Spiller, Brain, Sept., 1925.)

tion of which appeared to be within the brain-stem. In brief, he had perfect auditory function and no vestibular function.

Eleven days later he died. Respiration became rapid, shallow and irregular. Intellect remained keen. He suddenly became comatose and cyanosed, and died of respiratory failure. The pulse had been of comparatively good quality.

Fig. 3. (Section 33). The degeneration on each side of the raphé has united and implicated the raphé. (From Jones and Spiller, Brain, Sept., 1925.)

Autopsy negative in all details. The whole brain was removed and sent to Dr. William G. Spiller, who made the

exhaustive study. For our purposes the following gives the essence of his findings.

* * * * *

VIII NERVE.

The system of the VIII Nerve is one of the most complex and difficult to determine in the entire nervous system. The

Fig. 4. (Section 70). The degenerated area destroys all fibers at the center of the raphé. (From Jones and Spiller, Brain, Sept., 1925.)

fibers of the VIII Nerve itself are well understood and also the pathways from the cochlea in the brain-stem. However,

knowledge of the vestibular pathways in the brain-stem had been obscure up to the time of this study.

The brain-stem from the lower portion of the medulla oblongata to the end of the aqueduct of Sylvius was cut in microscopic serial sections, over 1,000 in number, and of these a very large number was stained, every third or fourth section at some levels, by various methods.

Fig. 5. (Section 110). The central area of degeneration has greatly diminished in size at this level—corresponding to the nucleus of the nervus abducens on each side. (From Jones and Spiller, Brain, Sept., 1925.)

The important finding was a limited area of complete degeneration in the center of the tegmentum, extending from the

lower part of the medulla oblongata well up into the lower part of the pons, on each side of the raphé.

It may be concluded from this study that all or nearly all of the central tracts concerned with vestibular function pass through the area represented by this degeneration; also that few if any of the fibers of the cochlear system are represented in this region. The fibers of both the vestibular and cochlear portions of the VIII Nerve escaped entirely. In this case, although there was a complete vestibular block, the degeneration involved only the *central* tracts of the vestibular system.

Important changes in the form and number of nerve cells were found in the primary nuclei and it was concluded that the fibers from these nuclei extended into this central area of degeneration.

* * * * *

In the non-neurologic language of football, the Central Vestibular fibers (see Figs. 2, 3, 4 and 5) "buck the line" outside of tackle, in the medulla; then, higher up, between tackle and guard; and higher up, through center — whereas the auditory fibers run around the ends.

In streptomycin poisoning almost every symptom and every finding points to brain-stem lesion; yet in reports of these cases we find such terms as "aural disturbances," "otic complications," "labyrinthine functional upsets" and "reaction on the VIII Nerve apparatus" — all of which are not only vague but refer to the end-organ. Just as mumps selects the internal ear and the testicle, so streptomycin seems to have the equally strange selective affinity for this Central Vestibular Tract. The following (see Fig. 6) is identical with the classic findings of Dr. Spiller: A man, aged 57, had received streptomycin for bacterial endocarditis. Then came the familiar vestibular symptoms. His cochleae were unaffected. On looking up, there was a lateral wavering of the eyes. Turning produced no nystagmus, no motion-sensing and no constitutional responses. Even when he was turned with the head forward and then sat up there was no falling response whatever. After four minutes of douching of each ear there was no response

in nystagmus, motion-sensing, past-pointing or falling. Auditory function, unaffected. Vestibular function, nil.

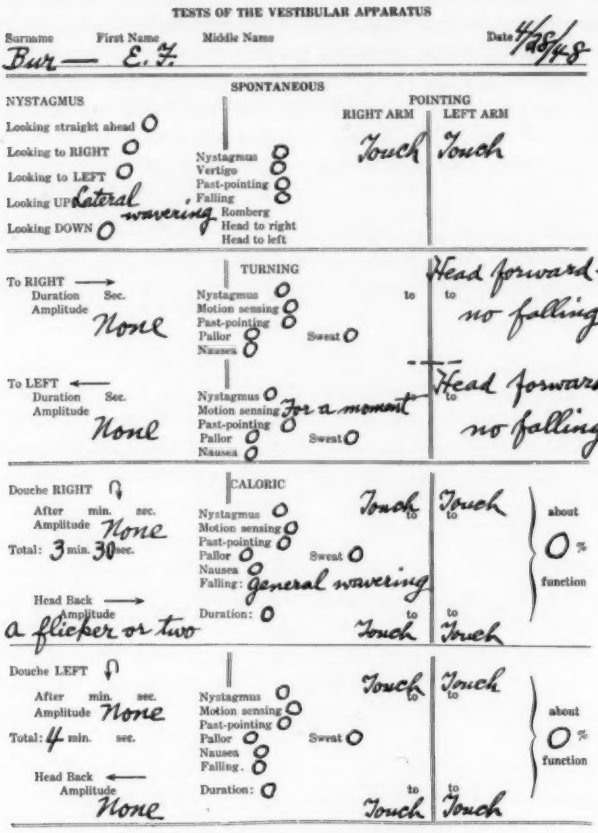

Fig. 6. Streptomycin poisoning.

Conclusion: In streptomycin poisoning other structures may also be involved, but the essential lesion is in the Central Vestibular Tract.

635 South Westlake Avenue.

PARALYSIS OF THE LARYNX FOLLOWING A PROPHYLACTIC INJECTION OF TETANUS ANTITOXIN.*

HENRY DINTENFASS, M.D.,
Philadelphia, Pa.

Complications following the use of serum both for prophylaxis and in the treatment of disease are more common than is generally supposed.[1] This is especially true of tetanus antitoxin. The pathologic changes occuring are usually neurologic in character and may involve nerve structures anywhere in the body. A review of the literature has disclosed that the brachial plexus,[2] the oculomotor,[3] the facial,[1] the auditory[4] and the recurrent laryngeal nerves[5] have been most often affected either singly or in combination. Imperatori, Neffson, Floyd, *et al.*, and Clerf have had experiences in which the recurrent nerve alone was involved. In the case to be presented, the recurrent nerve was involved simultaneously with the brachial plexus.

M. R., a white, 33-year-old male, sustained an injury to the palm of his left hand on July 1, 1948. The wound was properly cleansed and adequate antisepsis used. The next day 1,500 units of tetanus antitoxin were injected into his left arm after sensitivity tests of the skin had given negative results. In the succeeding week he complained of constant nausea with general bodily weakness especially marked in both upper extremities. There was considerable pain in the shoulders, which radiated to the back of the neck. A tender lymphatic gland had developed in the left axilla. On July 9, he received penicillin in oil, 300,000 units, intramuscularly. Twenty-four hours later, several large hives appeared on the left arm, and adrenalin hypodermically was administered. When he awakened on the morning of July 13, he found he was unable to talk above a whisper. The weakness of the right upper extremity had cleared, but on the left side it had remained.

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I first saw the patient on July 19. He was completely aphonic and had been so for the preceding week. His previous medical history was irrelevant. The tonsils had already been removed. The sinuses were essentially negative for any pathology. The laryngoscopic examination disclosed the vocal cords to be in abduction, with loss of tension. When phonation was attempted, the cords showed no tendency to come toward the midline. The arytenoids were separated. There was a slight, ineffective cough present, significant of a lack of expulsive air power because of the failure of the glottis to close. His most strenuous efforts to talk resulted in a forced whisper. Recurrent nerve involvement with paralysis of the larynx was diagnosed. Movements of the left shoulder at this time gave evidence of weakness. He raised the left hand to the level of the head with difficulty and then could hold it in that position for only a short while. There was thinning of the musculature over the left scapula.

Three weks following his injury, the weakness of the left shoulder had become more pronounced, and atrophy of the left scapula muscles was quite apparent. The laryngeal picture remained exactly the same until Sept. 19, *i.e.*, about nine weeks after the onset of his trouble, when beginning vocal cord movements were observed. In the next few days definite adduction was noticeable and with this a slight return of the voice. The tone at first was harsh, but in a period of a week improvement was so rapid that there was good phonation with very little effort. At the present writing, five months after his accident, although the voice quality is good, there is atrophy of the muscles of the left arm and shoulder, in addition to the involvement of the scapula muscles.

The causative factor of the laryngeal paralysis here was unquestionably the serum injection. This is well proven by the case history, the reports in the literature and the associated paralysis of the brachial plexus. The diagnosis is to be differentiated from paralysis of the vocal cords or functional origin. Functional paralysis is usually brought on by a sudden shock, scare or overuse of the voice and is frequently seen in hysterical females. Muscular atrophy is never present.

This case reveals the possible dangers ensuing from a supposedly harmless medical procedure. Fournot,[6] Gammelgaut[6] and others have reported fatalities after the administration of tetanus antitoxin given only for prophylactic purposes. Regarding the pathologic processes involved, Kraus and Chaney[7] believe that a disorder of the blood vessels occurs, causing nutritive impairment of the tissues of the nervous system and interfering for a time with the activity of the nerve fibres and cells, but occasionally producing cell death and parenchymal necrosis. This theory is well illustrated in our patient and explains why the laryngeal paralysis was of a temporary nature, whereas the brachial plexus involvement resulted in atrophy of the shoulder girdle muscles.

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1305 Spruce Street.

VIRUSES IN THE OROPHARYNX AND NASAL PASSAGES.*

JOHN F. ENDERS, Ph.D.† (by invitation),
Boston, Mass.

It is somewhat difficult to select from the now very large body of information concerning viruses those aspects which might be of especial interest to members of this group. After weighing this problem of selection, it seemed perhaps most profitable to review rapidly those viruses pathogenic for man which, during the course of the disease they incite, may be present in the mouth, pharynx and adjacent structures; then, with a general survey of this sort completed, to consider in more detail our present knowledge of a few of the virus diseases which the otolaryngologist may encounter.

I have decided on this procedure not merely because I hope that the review of the factual material may be helpful, but also to call your attention to two misconceptions which, as far as I can judge, are somewhat widespread. Both of them have arisen, naturally enough, from the rapid advances that have been made during the last 15 years in our knowledge of viruses. Within this period a considerable number of agents of this class have been recognized as the cause of disease in man, and new techniques for the laboratory diagnosis of many viral infections have been developed. Because of these advances two inferences have been drawn: *1.* laboratories equipped for the study of viruses are prepared to attempt

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†Chief, Research Division of Infectious Diseases, the Children's Medical Center, Children's Hospital, Boston, and Associate Professor of Bacteriology and Immunology, Harvard Medical School.

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the isolation of virus or to carry out diagnostic serologic tests on any specimen which may be submitted with a request for "virus studies"; *2.* a tentative diagnosis of "virus infection" is appropriate in conditions which appear to be infectious but in which no etiologic agent can be identified on the basis of either clinical or laboratory data. That both these inferences are unwarranted will, I trust, appear from the remarks which follow.

We may turn now to a summary of those viruses which attack man and which appear in the nose, the throat or the mouth during the course of the infection. In Table 1, I have attempted to list as completely as possible the infections of this sort in which there is definite evidence that the virus is present in the sites I have just mentioned. This evidence consists either in the isolation of the agent from saliva, nasal discharges or nasopharyngeal washings, or in epidemiologic observations which appear to be conclusive. The table also indicates whether or not practical methods are available for the isolation of the virus or its identification as the infecting agent by serologic techniques.

The tabulation reveals three points of interest. In the first place it shows that the number of viruses which may be found in the nose, mouth or throat is considerable. Indeed, their number certainly equals if it does not exceed that of the various species of pathogenic bacteria which may invade these structures.

A priori this fact suggests that the cells of the mouth and upper respiratory tract provide favorable conditions for the multiplication of many viruses. In certain instances, such as herpes simplex stomatitis, herpes zoster, influenza, common colds, varicella, smallpox, measles and foot-and-mouth disease and the stomatitis and diarrhea of infants, this hypothesis is supported by the direct evidence of localized tissue injury — injury that is presumably the result of the local proliferation of the virus. On the other hand, there is little doubt that in rabies and mumps all the virus present in the saliva arises either in the parotid gland or its associated nervous connec-

TABLE 1. VIRUS DISEASES IN WHICH AGENT MAY BE PRESENT IN OROPHARYNX OR NASAL PASSAGES.

Tissues Most Frequently Involved			
Skin	Respiratory or Gastrointestinal Tract	CNS	Others
*Herpes simplex	*Epidemic influenza	*Rabies	*Mumps
*Smallpox	†Infantile stomatitis and diarrhea	*Lymphocytic choriomenin.	
*Foot-and-mouth disease	‡Common colds	*Russian Far East enceph.	
†Measles	‡Primary atypical pneumonia	*Venezuelan equine enceph.	
‡German measles		*Japanese B enceph. (?)	
‡Chickenpox		†Poliomyelitis	
‡Herpes zoster			

*Methods for isolation of virus available. Diagnosis possible by tests for specific antibody.
†Methods for isolation of virus complex and uncertain. No tests for specific antibody.
‡No methods for isolation of virus. No tests for specific antibody.

tions. Between these two groups in which the situation is reasonably clear lie the virus diseases of the central nervous system other than rabies which are listed in the table. Here, in the absence of clearly defined pathologic changes, we are quite uncertain as to whether the agents multiply locally, or are transported to the mucous membranes via the blood, the lymph or the nervous pathways from peripheral ganglia or the central nervous system itself. In the case of poliomyelitis, the virus has been isolated from the tonsil, but this does not prove that proliferation in this tissue has occurred. Probably within the near future, by means of tissue culture methods which are now available, it may be directly shown whether or not the virus of poliomyelitis as well as the other neutropic agents can mulitply within cells of the tonsil, as well as of other tissues derived from the oropharynx.

With possibly a few doubtful exceptions the table, as I have said, defines the limits of the group of known viruses which occur in the areas under consideration. Future investigations may reveal the etiologic rôle of as yet unknown filtrable agents in certain conditions whose causes are now obscure. Such research may also demonstrate some hitherto unsuspected effect of the viruses that we already recognize. At present, however, it would seem to be the part of wisdom and good science to preserve a rigorously critical attitude when it is vaguely proposed that a "virus" is responsible for a lesion of uncertain origin.

This statement, however, does not imply that in certain diseases in which the etiology is undefined the possible participation of a virus should not be entertained. Suggestions such as that made recently by H. G. Tobey[1] that a virus may be involved in cases of otitis media in which the effusion is bacteriologically sterile deserve to be seriously considered and followed up by experimental study. Indeed, it is often the observant clinician who stimulates the laboratory worker to renewed etiologic research.

Finally, Table 1, with its footnotes, indicates those infections in which it is possible by laboratory procedures to confirm or establish the diagnosis. It may be a satisfaction to

investigators in the virus field that in nine of these 17 diseases such methods have been made available; nevertheless, their impracticality in the routine diagnosis of a disease as important as nonparalytic poliomyelitis as well as their complete deficiency in most of the common viral diseases of childhood must be regarded as unsatisfactory.

When the laboratory diagnosis can be made, it is based either upon the isolation of the virus in a susceptible animal or upon the demonstration of the development of specific antibody during the acute phase and early convalescence. Even with those viruses that are most easily propagated in the laboratory, such as influenza, mumps and herpes simplex, the procedures are time-consuming and expensive. Accordingly, this mode of diagnosis should be in most instances employed only under special circumstances, as in the determination of the prevailing agent in epidemics or when the clinical data strongly suggest the nature of the infection.

In contrast to isolation of the virus, serologic tests provide a relatively convenient and rapid means of diagnosis, especially when the techniques of complement fixation or the inhibition of virus hemagglutination are employed. If it is necessary to depend upon the demonstration of the development of virus-neutralizing or protective antibodies, the procedure requires much more time. Fortunately, the complement fixation reaction has now been found to be applicable to most of the viruses which can be maintained in the laboratory; moreover, infections with the viruses of mumps and influenza can be readily distinguished not only by the complement fiaxation test but also by the red cell inhibition reaction which I have mentioned. In all serologic tests it is necessary for a final diagnosis to obtain two specimens of serum, one as early as possible in the acute phase of the disease and another from one to three weeks later. When these are tested simultaneously, any significant increase in specific antibody which occurred during the interval is revealed.

Although comparatively easy to carry out, these tests, with the exception of that for inhibition of hemagglutination, require considerable labor by experienced personnel. Requests

for their performance, therefore, should not be made unless the results are considered essential to the final estimate of the case. In this connection it should also be pointed out that the serologic findings nearly always provide a diagnosis only in retrospect, since recovery frequently occurs before they can obtained.

I shall now recapitulate very briefly a few of the more recent advances in knowledge of three diseases which may be of interest to members of this group. The disorders I have chosen are acute herpetic gingivostomatitis, stomatitis and diarrhea of infants, and mumps.

Acute herpetic gingivostomatitis was often formerly designated as aphthous stomatitis or Vincent's stomatitis. The etiologic agent until a few years ago was unknown, but many had believed that the fusospirochetal organisms of Vincent were responsible. In 1939, Dodd, Buddingh and Johnston[2] isolated the virus of herpes simplex from 27 of 28 cases of gingivostomatitis — a result which strongly suggested that this agent was actually the cause of the disease. Since herpes virus, however, may frequently be isolated from the saliva of normal persons, the evidence was not complete until Burnet and Williams[3] showed that virus-neutralizing antibodies developed in the blood during the course of the illness.

On the basis of these observations and the epidemiologic studies carried out by Burnet and his co-workers at about the same time, the condition is now generally regarded as a primary infection by the herpes virus. Occurring most often in young children, but occasionally in adults, this initial attack is characterized by high fever and enlargement of the regional lymph glands, in addition to the distinctive reddening of the gums and the formation of vesicles on the buccal mucosa, the tongue and, in some cases, the tonsil and pharynx.

Persons who have previously been infected with herpes virus and who continue to harbor it in their tissues also may develop stomatitis. This condition is now described as recurrent herpetic stomatitis. Scott, Steigman and Convey[4] have pointed out that in such cases the local lesions tend to be less

severe and the generalized disturbances are very mild or absent. Repeated attacks are common at any age. Since individuals who constantly harbor the virus maintain a high level of antibody in the blood, no change in its concentration can be usually demonstrated as a result of an attack.

In respect to these herpetic infections of the oropharynx, it is important to note in passing that Vincent's angina of the tonsil, for which the virus of herpes is not responsible, can, as Steigman and Scott[5] have recently shown, be easily distinguished from herpetic lesions of this organ.

From the foregoing remarks it is apparent that in order to obtain complete evidence of primary herpetic gingivostomatitis it would be necessary to demonstrate the virus in the saliva or materials from the lesions, as well as the development of virus-neutralizing antibody during the course of the infection. In practice the distinctive clinical picture, together with a positive serologic test, should be sufficient. Laboratory diagnosis of recurrent herpetic stomatitis would depend upon the isolation of the virus in the presence of elevated and unchanging neutralizing antibody and perhaps the occurrence of typical intranuclear inclusions in the cells obtained from the lesions.

Pediatricians have lately given much attention to another form of stomatitis which occurs predominantly in infants under six months of age and which is associated with diarrhea of varying severity. This condition was first described by Buddingh and Dodd,[6] in 1944, who obtained evidence that a filtrable agent exhibiting certain similarities to herpes simplex was involved.

Clinically, the disease is characterized by a sudden onset. Vesicles appear usually on the tip and under surface of the tongue and less often on the gums and inner surface of the lips. These lesions may persist from seven to 14 days. In the very young infant the accompanying diarrhea, which may frequently prove fatal, is often the predominating feature, whereas the stomatitis may be so mild as not to be observed. Older children and adults in contact with such cases may

develop stomatitis without diarrhea. It is probable that these individuals may transmit the disease to infants, although infection of the latter may also occur in the birth canal, since the virus has been demonstrated in the vaginal secretions of mothers whose children develop the disease.

By inoculating the cornea of rabbits with swabbings from the mouth lesions or with stools, Buddingh and Dodd produced characteristic lesions. By serial passage of emulsified infected cornea in these animals the virus has been maintained in the laboratory. Bacteria-free filtrates of infected corneal suspensions likewise produced the characteristic lesions. The development of virus neutralizing antibody during the course of the illness in infants was also revealed.

Although Buddingh and Dodd, as well as other workers, have not always been able to repeat these experiments, there can be little doubt that a virus is the cause of this type of infant diarrhea. In view of the great importance of the general problem of diarrhea of the newborn both in this country and in Europe, it is desirable that all those who frequently inspect the oropharynx should be acquainted with the observations of Buddingh and Dodd. By bringing cases of the sort they have described to the attention of those interested in the disease from the experimental point of view, they can aid materially in advancing knowledge of this important subject.

I have included the mumps virus in this discussion because it is one of the causes of deafness. Much progress has been made in our understanding of mumps infections during the past 15 years.[7] Within this period the etiologic agent has been definitely established as a filtrable virus, methods of propagating it in chick embryos and tissue cultures have been developed, as well as serologic techniques for its identification, which have proved particularly useful in the diagnosis where parotitis is not a prominent feature or cannot be observed, such as in certain cases of meningoencephalitis.

By means of one of the serologic procedures like the complement fixation test it has been possible to show that inapparent infections by this virus frequently occurs. Since

there are good grounds for believing that such inapparent or subclinical infections establish an immunity as complete and durable as the overt disease itself, the test, when positive, indicates not only past infection but also that the individual is in nearly all instances immune to re-exposure. The same information can also be obtained by inoculating a small quantity of the inactivated mumps virus into the skin. In a large proportion of persons who have previously been infected, a hypersensitive reaction marked by erythema and a variable degree of induration develops after 24 to 48 hours at the site of injection. In most individuals who have not been infected, little or no reaction is observed. These two methods—the complement fixation and skin tests—may be employed to confirm a positive history of mumps or to obtain information concerning the occurrence of past infection when the history is doubtful or negative. Accordingly, it would seem possible that in certain cases of deafness in which the cause is obscure, but in which the participation of the mumps virus is considered, these tests might prove helpful if not conclusive.

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PERSISTENT CRANIOPHARYNGEAL CANAL IN A LIVING ADULT.*

WILLIAM WESLEY WILKERSON, JR., M.D., and
LEE FARRAR CAYCE, M.D.,
Nashville, Tenn.

A peristent craniopharyngeal canal is an embryological anomaly which consists of an incomplete or a complete canal extending from the pharynx or from a blind end canal within the vomer through the septal area of the sphenoid sinuses to the hypophysis. From the study of anatomical specimens this rare anomaly has been found to end distally in the vomer in a high percentage of cases, as would be indicated by the embryology of this area.

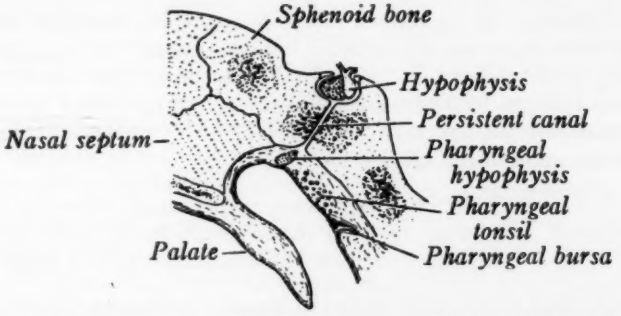

Fig. 1. Relations of the hypophysis and pharyngeal hypophysis of the newborn, showing in median section (×1). (From Arey's "Developmental Anatomy" after Frazer. W. B. Saunders Co.)

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An extensive search of the literature has revealed no report of a case in which a craniopharyngeal canal was noted in a living human. It is our belief, therefore, that this essay deals with the first reported case of a craniopharyngeal canal in a living person and is all the more rare because it was found in an adult.

PLATE 2.

Name of Investigator	Newborn	Per Cent
Le Double[23]	More common in boys	0.09
	Apes	30
	Rabbits	100

The incidence of the craniopharyngeal canal in the human, both in infancy and adulthood, and in other animals, is shown in these slides. This is a composite tabulation comprising the results of many investigators. From these statistics we note that a persistent craniopharyngeal canal in the human is neither atavistic nor directly inherited.

HISTORY OF THE CRANIOPHARYNGEAL CANAL.

Rathke,[33] the discoverer of the pouch which bears his name, mentioned a case of cerebral hernia through an opening in the sphenoid bone, in 1839. Rathke assumed that this hernia was induced by hydrocephalus which had developed prior to closure of the aperture through which the so-called Rathke's pouch entered the cranial cavity.

Other articles on craniopharyngeal canals were written between 1839 and 1875 by Laurence,[22] Houel,[16] Wegelin,[51] Wallman,[50] Rippmann,[35], von Mihalkovics[48] and Landzert.[21]

In 1878, Kulischer[20] observed a case in which hydrocephalus in the embryonal stage induced cerebral hernia through the craniopharyngeal canal. In 1879, Parker and Bettany[29] found that the craniopharyngeal canal was always present in rabbits.

In 1882, Froriep[12] mentioned the craniopharyngeal canal as having been determined in human embryos during the first three months.

Description of the craniopharyngeal canal in the macerated human skull was published for the first time in 1886, by Romiti.[37] He had examined about 800 crania and had found the craniopharyngeal canal only once in the skull of a child, five years old. This skull was well developed and brachiocephalic. On the under surface of the sphenoid bone, somewhat to the right of the median line, 6 mm. from the sphenooccipital synchondrosis and 3 mm. from the upper end of the vomer, there was an opening 1 mm. in diameter. It was the inferior opening of the craniopharyngeal canal, which in length measured 8 mm. The superior opening lay in the region of the sella turcica, 2.5 mm. to the right of the median line.

PLATE 3. INCIDENCE OF CRANIOPHARYNGEAL CANAL

(Ancient Skulls and Cadavers).

Name of Investigator	Human	Per Cent	
Pende[30]	Human	0.3	
Rizzo[36]	Human	0.29	
Sokolow[39]	Human	0.3	(16 out of 5,281 adult skulls)
Le Double[23]	Human	0.20	
Rossi[38]	Human	0.24	(Examined 3,127 skulls)
Romiti[37]	Human	0.125	(Examined 800 skulls)
Average		0.2425	

In the following year, 1887, Suchannek[42] observed a case of persistence of the craniopharyngeal canal, in a four-year-old girl. His preparation showed a repression in the pharynx.

In the following year, 1887, Suchannek[42] observed a case of persistence of the craniopharyngeal canal, in a four-year-old girl. His preparation showed a depression in the pharyngeal roof, exactly opposite the fossa of the hypophysis. From this fossa, a mushroom-shaped, roundish formation of hypophyseal tissue protruded approximately 4 mm. and was 3.5 mm. in diameter. There was also a small button-shaped exostosis on the right posterior clinoid process. The brownish color of the hypophysis differentiated it from the covering of dura mater which immediately surrounded it and the adjoining bone lamella of the sphenoid body. The existence of the exos-

tosis on the right posterior clinoid process was explained by Suchannek[42] as a remnant of the middle trabecula of the cranium.

In 1890, Sternberg[41] described a craniopharyngeal canal which he observed in the skull of a child, three to four years old.

The Italians were assiduous in their research on this subject between 1890 and 1900. Maggi,[25] Rossi[38] and Rizzo[36] made extensive studies on human skulls.

Testut,[43] in 1893, stated, "One finds occasionally in newborn infants a canal which starts from the sella turcica and ends in the pharynx. This is the craniopharyngeal canal, well described by Landzert,[21] and enclosing with a few vessels a prolongation of the dura mater. Romiti[37] has just observed this canal in a cadaver of a small girl of five years."

Spee,[40] in 1896, reported: "As an extremely rare formation due to inhibition, we find in the floor of the hypophyseal fossa the persistence of a canal, the craniopharyngeal, which perforates the basal sphenoid, and is looked upon as the site of departure of the hypophysary duct, which has remained open. In the hare, this aperture is typical."

Rauber,[34] in 1902, discusses the upper orifice of this median canal in the pituitary fossa, which passes through the body of the sphenoid bone and terminates on its lower surface. He says: "It contains the duct of the hypophysis, but it must not be mistaken for the venous canals, which also occur. In this connection we should again note the fact that the upper orifice of the canal usually occurs in the lowest portion of the sella in the median plane."

It was first suggested by Mott and Barrett,[28] in 1899, that craniopharyngeomas were related to craniopharyngeal canals. This theory was proved true by Erdheim[9] in 1904.

There are few references to craniopharyngeal canals to be found in the literature of the twentieth century. Most of these articles deal with conditions in various mammals other than man. Barrier Gustave et Petit,[2] in 1908, and Zannini,[53] in 1922, wrote of the craniopharyngeal canal in the horse. The

latter article deals in an instructive manner with the human phase of this subject.

Levi,[24] in 1909, described a craniopharyngeal canal in an acromeglia.

Sokolow,[39] Waldeyer,[49] Cave,[5] Dursy,[8] Arai[1] and Mannu[26] reported the characteristics, frequency, etc., of the craniopharyngeal canal in fish, reptiles and mammals other than human.

EMBRYOLOGY.

In a 7 mm. embryo the telencephalic portion of the forebrain has grown laterally and forward. Rathke's[33] pouch at this stage is merely a depression formed by stresses of the surrounding structures with the forewall of the pouch being composed of ectoderm pushing against the posterior wall of the forebrain.

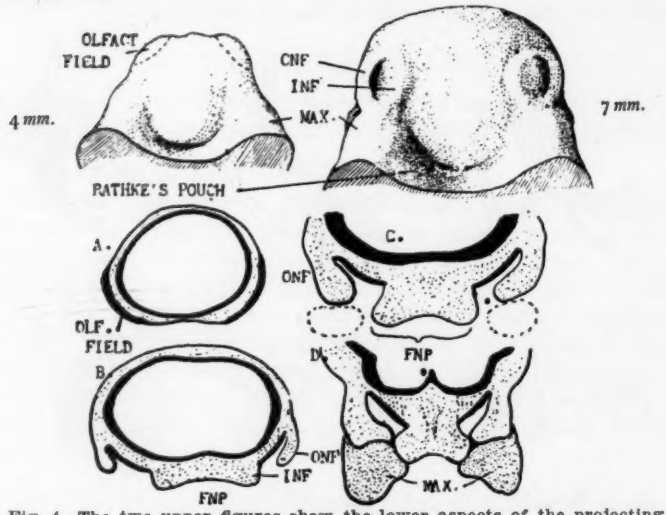

Fig. 4. The two upper figures show the lower aspects of the projecting heads of embryos of 4 and 7 mm. In the 7 mm. specimen the growth of the telencephalic part of the forebrain, increasing in size laterally as well as forward, has brought the olfactory fields below the head, instead of at its anterolateral aspect, as in the 4 mm. embryo. Outlines of sections through these two heads are given in (A) and (B), below and on the left. These show formation of olfactory pits and frontonasal process (FNP). Diagrams of further changes are given in (C) and (D). The maxillary processes are indicated by interrupted lines in (C), showing where they will come into position, as in (D). (ONF), (INF), outer or lateral and inner or medial nasal folds. (MAX), maxillary process (from "Buchanan's Anatomy.") From Frazer's "A Manual of Embryology," Williams and Wilkins Co.

Rathke's[33] pouch lies between the bucopharyngeal membrane and the forebrain with the maxillary processes exerting influence from their lateral positions.

The mesodermal thickening causes the pouch to elongate. At this stage the roof of the pharynx is the canal's upper border. The growth of the sphenoid bone compresses the neck of the pouch so that by the 28 mm. to 36 mm. stage only the canal proper remains.

The canal proper, that portion of this ectodermal elongation connecting the base of the brain and the roof of the mouth, is encroached upon by the septal processes of the maxillary mesoderm in forming the nasal septum.

A craniopharyngeal canal may, therefore, extend into the nasal septum in case the septal processes fail to compress the anterior end of this pouch by the 20 mm. stage.

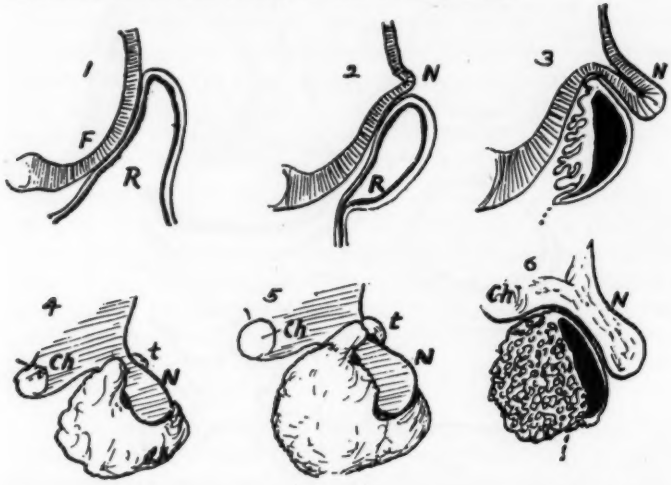

Fig. 5. (Fig. 173—To show formation of pituitary.) (R) is the wide'y open (1) pouch closely applied to the forebrain (F). In (2) its separation is beginning below, so that proliferation of its front wall is possib e here. (3) A similar process occurs laterally. At the same time the neural lobe (N) grows out from the forebrain and forms a club-shaped process above and behind the pouch. In (4) the completely separated pouch is forming by proliferation; the anterolateral lobes and corneal upgrowths (t) begin to surround the neck of the neural lobe, to form the pars tuberalis (5). The neural lobe lies on the posterior wall of the pouch (6), which is the pars intermedia; a transverse section would show this curved around the anterior and lateral aspects of the pars neuralis. (Ch) Chiasma.

The cranial portion of Rathke's pouch dilates and forms the anterior lobe of the hypophysis. The fact that such a development occurs from this canal explains the reason pituitary rests are found in the pharynx and in a persistent craniopharyngeal canal.

PLATE 6. ESTIMATED TIME OF CLOSURE OF RATHKE'S POUCH.

Tourneux[45]	Beginning of third fetal month
Hertwig[15]	Shortly before birth
Pende[30]	Beginning of third fetal month
Levi[24]	Between second and third fetal month
Le Double[23]	Excessively rare one month after birth
Escat[10]	Occasionally after birth
Landzert[21]	

Table 6. (From Frazier's "A Manual of Embryology," Williams and Wilkins Co.

DESCRIPTION WITH DISCUSSION.

The description of the craniopharyngeal canal, rarely found in the living, is derived from embryological and anatomical studies.

The canal, according to Mannu,[26] has an average length of 2.5 cm. The internal orifice, situated anteriorly in the hypophyseal fossa, is either round or elliptical in shape. The external orifice is of the same shape, although larger, and is 1 to 2 mm. from the vomer. The external opening is located on the face of the sphenoid bone, usually in the intraseptal region. Mannu found in his three cases that the canals were not uniform in calibre, being narrower in the midportion. He further stated that the canals permitted the passage of a medium sized horse hair.

Cave[5] found a craniopharyngeal canal in a skull of a 38-year-old man with an internal orifice 3.5 mm. in diameter. A probe introduced into this orifice was passed downward and forward for 16 mm. through the medial septum of the sphenoid sinus to a vomerine termination in the nasal septum. Concomitant departures from the normal were manifested in the same subject.

Fig. I

Fig. II

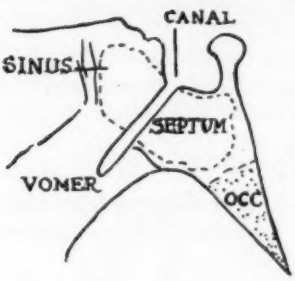

Fig. III

Fig. IV

Fig. 7. Presents a circular hole, 3.5 mm. in diameter, the mouth of the craniopharyngeal canal, through which a probe can be passed 16 mm. downward and forward through the medium septum of the sphenoid sinuses to a vomerine termination. Vascular foramen are also seen. Other abnormalities are noticeable.

The transverse bar of the dorsum sella is attached to the main body of the bone by fibrous tissue only.

The relations of the canal (from skiagram).

Shows enlargement of a vertebral body indicative of increased pituitary activity similar to that noted in acromegalia.

(From Cave's "The Craniopharyngeal Canal in Man and Anthropoids," Jour. Anat., Vol. 65, 1931.)

Tweedie and Keith[46] reported a case in which the pituitary body was located in a cleft of the palate. This case had a harelip and a groove on the columella of the nose. The mass extended from the roof of the nasopharynx and, therefore, was a herniation through the craniopharyngeal canal. Six other cases of herniation have been reported.

Waldeyer[49] in his study of craniopharyngeal canals also found communications between the cavity of the sphenoid sinus, through the inferior wall and the pharynx to be fairly common. Undoubtedly these openings may be a considerable factor in the transmission of infections from the pharynx to the sphenoid sinus and vice versa.

Citella[6] found, in addition to the habitual prolongation of the dura mater, several small blood vessels which connected with those of the pharyngeal vault. When he injected these vessels with India ink at the fundus of the sella turcica, the superficial and deep vessels of the pharyngeal vault were deeply stained. This is, therefore, an unusual pathway of intracranial infections originating in the hyperpharynx vault.

The contents of the canal have been described, as a single fibrous strand by Prenant[31] and Landzert,[21] as connective tissue and blood vessels by Waldeyer[49] and Mingazzini,[27] as a prolongation of the dura with blood vessels by Citelli,[6] Sokolow,[39], Testut[43] and W. Krause,[19] or as perichondrium by Froriep.[12]

Von Luschka,[47] Froriep[12] and Suchannek[42] found hypophyseal rests in the canal. Arai[1] found a true accessory hypophysis in the canal. Haberfeld[14] and Calori[4] found craniopharyngeal canals filled with pituitary glandular tissue which protruded into the hyperpharynx and could have been mistaken for polypi or adenoid tissue.

Killian,[18] in 1888, stated that nests of pituitary tissue may occur in the nasopharynx. Later, Haberfeld[14] and others found these nests of pituitary tissue to be a constant finding in the nasopharynx of the human.

CRANIOPHARYNGIOMAS.

Craniopharyngiomas, which are encapsulated squamous epithelial tumors, can be divided into two groups, the solid and the cystic types. The latter predominates in a large series of cases. The solid tumors commonly are nonmalignant. Malignancy in this area usually arises from within the nasopharynx.

Craniopharyngiomas are more often seen in children, constituting 10 to 13 per cent of all intracranial new growths in childhood. These tumors, however, have occurred at varying ages, four cases having been reported in patients over 60 years of age.

Haberfeld's[14] article on new growths which develop from hypophyseal rests is elucidating.

In an article by Ingraham and Scott[17] it was stated that, "Erdheim[9] had pointed out that in the embryonic development of the hypophysis from a union of infundibular and buccal pouch components, the epithelium comprising the original forward wall of Rathke's[33] pouch comes eventually to form the anterosuperior covering of the anterior lobe of the hypophysis. Squamous cell rests occupying this location have been observed repeatedly in autopsy studies in the absence of neoplasm. Proliferation of these squamous epithelial cells results in the epithelial masses with cyst formation that are known as suprasellar cysts, hypophyseal duct tumors, adamantinomas and craniopharyngiomas. Cushing[7] coined the term 'craniopharyngioma' because he felt it was accurately descriptive of the tumor's unique origin. Bremer[3] recently pointed out that from the embryological point of view this widely accepted term is not accurate, since Rathke's[33] pouch is a diverticulum of the buccal cavity and not of the embryonic pharynx."

Only a superficial general discussion of the pathology of craniopharyngiomas is indicated in this review. An hypopyseal neoplasm may be a small cyst or a large multiloculated cyst with islands of solid squamous epithelial tissue. It is in these solid masses that calcareus deposits are found, particularly at the base of the mass near its attachment where there is ample blood supply. These calcium deposits, when seen in a Roentgenogram, are pathognomonic of a craniopharyngiomatous tumor. The contents of the cyst are degenerated, desquamated, liquefied epithelial cells of a brownish color. Cholesterol crystals are found in this liquid, floating on the surface.

The solid tumors, adamantinomas, are epithelial masses in which no cells or debris have undergone liquefaction.

The consensus of opinion indicates that intracranial surgery for craniopharyngioma is the treatment of choice despite almost 100 per cent mortality, which results from the difficulty in entirely extirpating the growth. Intracranial surgery, however, serves to lengthen life and prevent blindness for a period of time. When the mass is incompletely removed, the cystic material usually ceases to drain as the cyst becomes adherent to the arachnoid; however, should the mass be connected with the ventricular system, the drainage would continue. Results show that radiation is of little value; however, it was recommended by Frazer.[11]

Although certain anatomical considerations and accepted surgical criteria would be violated by the opening of craniopharyngeal cysts through the nasopharynx, it is worthy of consideration, when possible, since the mortality rate of intracranial surgery is so very high. Tonnis[44] reports such a surgical procedure.

Globus and Gang[13] published a comprehensive article on "Craniopharyngioma" in 1945, an outline of which is listed below:

"*1.* Fourteen cases of hypophyseal duct tumor are described.

"*2.* The tumors were found to be derived from residues of the so-called craniopharyngeal (cranio-oral) duct.

"*3.* The tumors were subgrouped as cystic and solid; and in each subgroup there were those which were identified as papillomatous and those which were partially or dominantly adamantinomatous.

"*4.* The tumors often implicated the floor of the third ventricle, or broke through it to expand into the third ventricle, thereby causing hypothalmic disorders.

"*5.* Clinically, the recorded cases presented constellations of symptoms and signs which offered substantial diagnostic leads. Among the latter, in order of their significance and in varying degree, were:

"*a.* Vegetative disturbances, such as retarded growth, disturbance in fat distribution with occasional underdevelopment of secondary sex characteristics (Froelich's syndrome), imbalance in water metabolism (polydipsia and polyuria), amenorrhea, loss of libido, pulse and temperature discord, anorexia, obstinate constipation.

"*b.* General manifestation of increased intracranial tension, which included headache, accompanied by nausea and vomiting, papilledema and mental retardation.

"*c.* Signs due to direct pressure by tumor, optic atrophy, loss of acuity of vision, hemianopic field defects, pyramidal and cerebellar signs.

"*d.* X-ray findings, consisting of calcifications in or about the sella turcica.

"*6.* Surgical intervention in many instances resulted in but temporary relief and, at best, in a relatively longer survival period."

CASE REPORT.

J. F., W-F, age 19. The patient's only otolaryngological complaint was the inability to breathe through the left naris. Two previous surgical procedures had been but temporarily successful. The history indicated the earlier presence of sinusitis on the left side. The family history was negative.

The examination of the right naris was negative. The left naris was filled with a glassy mucoid secretion which was removed by suction. The floor of the nose was elevated, but only on the involved side. The entire nares were slightly congested, but no pus was seen. The anterior cervical and the submaxillary glands on the left side were moderately enlarged. The nasopharynx appeared to be normal; however, with a mirror the left choanae was seen to be occluded with what appeared to be a dense grayish membrane. The occluding wall protruded posteriorly in its central portion, and a ridge approximately 1 mm. thick was noted on its inferior border. With the use of probes a diagnosis of unilateral complete bony occlusion of the left choanae was made. A Roentgen examination revealed choanal atresia and also an increase in the density of the left frontal, ethmoid and maxillary sinuses. Other than that described above, the ear, nose and throat examination was negative. The general examination was negative with the exception of endocrine disturbances.

Under general anesthesia, both the posterior end of the septum and the occluding wall were removed. The technique of this surgical procedure, including the use of electrically driven burrs, was described by Wilkerson and Cayce[52] in a previous article, which included a brief report of this case.

While using a probe to determine whether or not the atretic wall had been completely removed, an irregular round opening was found in what we thought it to be the face of the left sphenoid sinus. This defect consisted of a canal beginning in the superior central portion of the anterior surface of the sphenoid and extending posteriorly superiorly to the sella turcica. The walls of the canal were of bone, overlying which a thin lining of soft tissue was noted during the examination of this area with a probe.

The orifice of the canal was estimated to be 3 to 5 mm. in diameter and the length 2 cm.

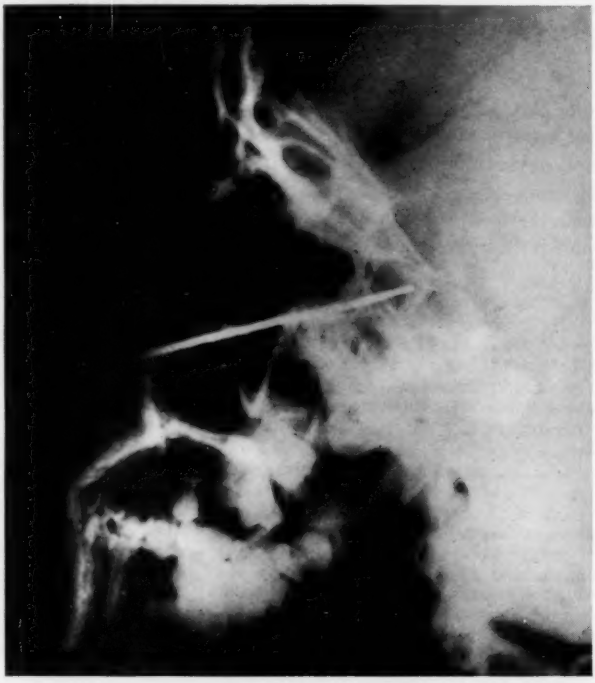

Fig. 8. Probe in persistent craniopharyngeal canal.

Many Roentgen examinations were made with probes inserted in the canal. Lipiodol was introduced into the sinuses several times, using the Proetz[32] method; however, the contrasting media at no time entered the canal, probaby due to a barrier of the soft tissue within the canal.

Two possible diagnoses were considered: a craniopharyngeal canal or a congenital dehiscence in the face of the sphenoid bone. The latter was disproved by probing both sphenoid sinuses extensively, during which

procedure the canal was located specifically within the septal wall lying between the cavities of the sphenoid sinuses. A definite diagnosis of craniopharyngeal canal was then established.

In view of the fact that the anterior lobe of the hypophysis is formed from the craniopharyngeal canal, it is interesting to note that this patient suffered from endocrine disturbances. Her internist found from a clinical examination, including a basal metabolic rate, that she suffered from hypothyroidism. At the age of 19, without pelvic infection or irradiation, she continued to have a secondary amenorrhea.

The current history of this patient has now been followed for two years since the atresial surgery. She has presented a consistently negative neurological picture.

SUMMARY.

Concomitant congenital defects are interesting in this particular case because of their rarity. The inhibition of embryological development, when resulting in a craniopharyngeal canal, may also, in some manner, cause a deficiency of the anterior lobe of the hypophysis, which probably accounts for the patient's hypothyroidism and secondary amenorrhea.

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POSTNASAL DISCHARGE.*

BEN L. BRYANT, M.D.,
Los Angeles, Calif.

What is the most common complaint voiced in the rhinologist's office today? Postnasal discharge — alone or in company with other related symptoms. This endeavor, therefore, is not to investigate the source and cause of some rare phenomenon; nor to present a novel method of combining and utilizing available therapeutic substances in fulminating diseases; nor yet to delineate a modification of a particular surgical procedure — it is an attempt to focus attention on a conservative manner of treating a remarkably frequent cause of this most common complaint, supported by microscopic studies of the tissues involved.

For years, it has been an observation of the author that in patients complaining of excessive postnasal discharge — with or without demonstrable sinus disease — there can be found, almost invariably, enlargement of the posterior tips of the inferior turbinates. This hypertrophy is not always visible on casual inspection of the nose, nor even by posterior rhinoscopy. It should be emphasized at once that sometimes this change can be visualized only by means of the nasopharyngoscope. In other instances, depending upon various factors, such as the degree of hypertrophy, the form of the turbinal body, and the architecture of the nose, the local change can be seen readily by anterior rhinoscopy, especially if it has reached the almost pendent "raspberry" stage. This type can

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, Calif., Jan. 29, 1949.

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scarcely be overlooked, but that which is not visible by anterior rhinoscopy — and which is found to be strikingly pronounced when viewed with the nasopharyngoscope—can be, and all too often is overlooked and receives no attention. This implies the importance — indeed, the necessity of including inspection with the nasopharyngoscope in every routine examination of the nose. If this is omitted, much valuable diagnostic information relative to the posterior part of the nose and the nasopharynx will be precluded.

The posterior tip hypertrophy or hyperplasia has been variously explained in the past. One popular notion holds that it results from the insult of purulent or mucopurulent drainage from diseased sinuses; ergo, that it does not occur without pre-existent sinus disease. This has been one of the factors leading to much of what most of us will agree has been poorly indicated, superfluous and unnecessarily radical sinus surgery in the past; and who can number the individuals who, after such surgery, continue to have excessive postnasal discharge as a result of the neglect of the posterior tip enlargement?

It occurred to me some years since that perhaps in many of these cases the cart and the horse had been interchanged, and that actually the purulent sinusitis was an end-result of a series of phenomena instituted by the hypertrophy of the posterior tips — and that it might be reversible.

This reasoning was prompted by the very many examples of postnasal drainage persisting after sinus surgery, which ceased when the neglected posterior tip hypertrophy had been adequately treated. It was supported by the very many examples of postnasal drainage which ceased without surgery, when the same treatment was used.

The further consideration and more careful examination of the posterior portion of the turbinates was rewarded, as I observed the consistency not only with which they were found to be hypertrophied in these cases, but also with which the postnasal discharge decreased and ultimately stopped as they were treated. All of this is even more striking when the symptoms and findings are unilateral — as has been the case repeatedly.

In many instances, X-ray evidence of sphenoethmoidal sinusitis has disappeared after the return to normal of the turbinal tips, with no treatment other than that directed to their hypertrophy. In other cases of purulent sphenoethmoiditis, treatment of the posterior sinuses, including the Proetz displacement therapy, has been effective only after the enlargement of the turbinate tips has resolved under local treatment; furthermore, maxillary sinusitis, which is certainly frequently merely a further extension of sphenoethmoiditis, has likewise repeatedly cleared, not from local treatment to the antrum but only when attention has been given to the turbinate tips.

There need be no wonder at these observations if the fundamental physiology of the nose and accessory sinuses is kept in mind. The two requisites for normal physiological function of this area are aeration and drainage. Normal drainage resulting from ciliary action is toward the nasopharynx. Hypertrophy of the posterior tips of the turbinates sufficient to interfere with or retard this drainage causes a damming up of the secretions and this sets up the vicious circle of consequent congestion of the tissues, further disturbance of ciliary function and further interference with drainage. (It is known that, in response to chronic irritation, large numbers of goblet cells make their appearance in the nasal mucosa, and it has been demonstrated in rabbits that this histopathological change can be produced by the mere mechanical plugging of the nose.) The retained secretions become an excellent culture medium for the growth of organisms which would otherwise be removed, and the ground is laid for eventual purulent sinusitis.

There is surely little argument about the advisability of correcting that type of nasal septum deviation which by impingement interferes with the proper aeration of or drainage from a sinus or sinuses, and surely surgical treatment of sinus disease in such a case, without attention to such a septum deviation, would be considered inadequate. Why, then, does not the same fundamental principle apply to obstructive posterior tip hypertrophy — particularly as it can, and often

does, interfere with the normal physiological function of *all* of the sinuses? Admittedly, ultraconservatism can, on occasion, be a radical form of treatment, but increasing attention to those therapeutic procedures which lead to a restoration of normal nasal physiology, in preference to mutilating surgery, will give to the next generation of rhinologists far fewer nasal cripples, such as we have inherited as discouraging monuments to the past era of radicalism.

It is difficult to determine a single cause of all posterior tip hypertrophy. Undoubtedly, some are caused by lack of attention to the sphenoethmoidal region during and following an ordinary upper respiratory infection. (How often do we learn from a patient that his postnasal discharge commenced after a head cold or an attack of influenza!) In many instances, however, the hypertrophy develops gradually due to passive congestion of the region as a result of posture. This accounts for the instances of unilateral changes in the patients who sleep on one side. We are all familiar with the statement: "My nose blocks at night; the right nostril if I lie on my right side, the left nostril if I lie on my left side."

Once the hypertrophy attains a certain degree, the aforementioned vicious circle is established, and it must be interrupted if the patient is to have relief.

In the stage preceding the development of purulent sinusitis, there may be for months or years persistent, regular, excessive postnasal discharge, with each day initiated by an hour or so of "hawking" and spitting, often accompanied by gagging and vomiting. Eventually, the mesopharynx and hyperpharynx present islands and bands of lymphoid hyperplasia. These patients complain of a continued sense of irritation in the throat and often of pain; and they are subject to repeated attacks of acute nasopharyngitis as a result of the chronically irritated pharynx plus the fact that the inspired air is funneled and concentrated, instead of being normally disseminated. Because of this, they are subjected to various forms of local treatment of the pharynx, but not until treatment is directed to the posterior tip hypertrophy do they ordinarily obtain lasting relief.

At this stage, no evidence of sinus disease can be found, and inspection with the nasopharyngoscope, after thorough nasal shrinkage, reveals the nasal passages and meati to be normal, but at the posterior end of the inferior turbinate there is a berry-like, relaxed, pendulous mass, often polypoid in appearance. It is frequently surrounded by a pool of thick viscous mucus, which may extend to and overlie the Eustachian tube orifice. Many times an edematous thickening is seen to extend up the lateral wall from the inferior to the middle turbinate, with enlargement also of its posterior tip.

Thus we understand the additional symptoms often present, namely, those of Eustachian salpingitis — intractable to inflation —and those of pain of sphenopalatine ganglion distribution. How often has it been possible — and how gratifying — to witness the complete cessation of such symptoms of months' or years' duration following the conservative treatment of the hypertrophied region — and nothing else!

How, then, shall we treat this hypertrophy of the posterior tips? Their surgical removal has had considerably fewer advocates as detailed knowledge of normal nasal physiology has increased. We do not wish to sacrifice any normal nasal membrane, and we certainly wish to avoid the crusting that has all too often in the past followed surgery in this area, particularly if part of the turbinate bone has been removed; but a manner of therapy is available which has none of these disadvantages of surgery, and which is, furthermore, bloodless and painless. It is phenolization of the hypertrophied tissue.

I have heard objections to this method of treatment in the past, and I shall hear them again — but not from the patients! The objections voiced by the physicians have invariably proved to be on the basis of a misunderstanding of what is being attempted, or of faulty technique.

The usual criticism, of course, is that ciliated mucosa is being destroyed. Obviously, it is our desire and aim to destroy that portion of the posterior aspect of the turbinate which remains enlarged when the rest of the nasal tissues are shrunken; and, if the technique of the treatment is proper, no more than this unwanted tissue is affected.

A thin, light, malleable piano-wire type of applicator is used for the anesthetizing agent, and it is wrapped very lightly, the cotton then being compressed so that it is flat when applied. Very little cotton is used, so that when it is wrapped on the applicator it is no thicker than paper, and is half the size of one's little fingernail.

First, the nose is sprayed with a 2 per cent solution of cocaine-hydrochloride. Then the cotton applicator described above, moistened with a 20 per cent solution of the same drug, and bent to conform to the nose being treated, is placed so that the cotton is in contact with the turbinate tip and left there for 10 minutes. This ordinarily affords adequate analgesia for phenolization. Actually the phenol anesthetizes the tissue in 10 seconds, as can readily be demonstrated.

For the application of the phenol, the amount of cotton on the end of the same applicator is scarcely the size of the head of an ordinary pin. To avoid contact with any normal nasal tissue, the applicator bearing the phenol is usually introduced parallel to the floor of the nose, and is turned laterally to reach the posterior tip of the turbinate when it reaches the proper depth.

The application of the chemical is precise and "to the point"; and the course of the treatment can be checked accurately at any time with the aid of the nasopharyngoscope. Although the number of treatments varies with the degree of the hypertrophy, it is striking to note how frequently marked symptomatic relief is reported after the first or second treatment, even though the symptoms have been present for years.

Ordinarily there is no reaction to the treatment, but should it occur, it is nothing more than a temporary accentuation of the pre-existent symptoms. Phenolization of a given area is ordinarily not repeated at intervals of less than a week.

One's impulse, all too often, is to end the applications of phenol when the subjective symptoms have ceased, but they should be continued until nasopharyngoscopic inspection proves that the hypertrophy has been entirely eradicated; otherwise there will be an eventual return of the symptoms,

discouraging both to the patient and to the doctor. When the required course of treatment, varying with the case, has been completed, it is well to advise the patient of the importance of attending to any future head cold which does not clear completely in a few days.

How does the phenol act on the hypertrophy? After 11 years of using this procedure, and from clinical observation, and careful visual inspection, I was convinced that not only was there no deleterious action on the nose, but that, on the contrary, the entire nasal economy was improved, evidenced by lessened congestion of the membranes, freer nasal passages and clear sinuses. It seemed advisable to determine what actually occurred in the tissue and to examine the end-result histologically; therefore, biopsy specimens were removed from hypertrophied posterior tips of inferior turbinates, using a small sharp-edged biting forceps. Such specimens were taken before any treatment was instituted; 15 minutes after; one hour after; 48 hours after; and one week after phenolization (in this case the third application); and in one case a week after the ninth treatment, when the turbinate appeared normal, the patient's symptoms were entirely relieved, and she was dismissed.

The specimens were placed in formalin immediately after removal and within a few hours were blocked and sectioned for microscopic study.

In a section of tissue removed from a hypertrophied posterior tip of the inferior turbinate before any treatment, the epithelium shows some desquamation and erosion. There were numerous goblet cells. The basal membrane varied considerably in thickness and was infiltrated with lymphocytes, which extend into the stroma. The stroma presented marked edema, and contained many prominent, dilated, congested vascular spaces. The glands were in a state of hypersecretion.

Fifteen mintues after treatment with phenol the section was the same except for the appearance of a few polymorphs, in addition to an apparent increase in small round cells.

One hour after phenolization the section shows a marked increase in inflammatory cells, which pack the vascular spaces (which are much smaller) and invade the basal membrane and the stroma. Most of the epithelium is markedly thinned.

A section of tissue removed two days after phenolization shows a transitional epithelial covering varying in thickness and without goblet cells. The edema is somewhat less, the vascular spaces smaller, and there are many lymphocytes and a few plasma cells and large mononuclear cells.

A section of tissue removed from a turbinate which had been treated three times with phenol, and was taken one week after the third treatment. The surface epithelium is distinct and contains many goblet cells. The stroma shows still less edema, and the lymphocytic infiltrate, particularly in the deeper portion, is less marked. The cavernous spaces and glands are prominent.

A section of tissue from the turbinate which had been phenolized nine times, and was taken a week after the ninth teatment bears stratified columnar ciliated epithelium with a normal content of goblet cells. The basal membrane is normal in appearance. The stroma is dense and practically free of edema, the cellular infiltrate is mild, and the vascular spaces and glands have a normal appearance.

It is my opinion that this last section is the complete answer to those who have feared that the form of treatment has a harmful effect on the tissue concerned. It is worth mentioning that I also removed a bit of tissue from a turbinate which had had 14(!) applications of phenol, and the pathologist's report states, "surface epithelium seems quite normal and is well preserved."

From this study, it seems that as a result of phenolization the surface membrane shows intermediate alteration, as would be expected, but the final state shows no discernible deviation from normal. This corresponds with my clinical observations of these cases over the past 11 years, for they never develop crusting or other evidence of perverted surface membrane physiology.

SUMMARY.

Further attention has been directed to a simple and conservative treatment of a too-frequently neglected area of the nose, which is a most common cause of postnasal discharge, altered nasal physiology and sinusitis. It has been observed clinically in thousands of cases, and it has now been proved microscopically that phenolization of obstructive hypertrophy, when meticulously performed, has no deleterious effect. The relief of symptoms following this treatment is striking, the gratitude of the patients is touching, and one's conscience is free of the onus of having performed unnecessary sinus surgery and perhaps of adding to the number of nasal cripples extant, which is already too great.

STATE UNIVERSITY OF IOWA COURSE IN CLINICAL AUDIOMETRY.

An intensive two weeks' course in Clinical Audiometry will be offered at the State University of Iowa, June 14 to 28, 1949. The course will be conducted daily from 8:00 to 5:00 and will include lectures on audiometry, lip-reading, speech training for the hard of hearing, psychological problems of the acoustically handicapped and clinical otolaryngology; laboratory work, including observation and participating in hearing testing and hearing aid fittings for clinical patients in the Department of Otolaryngology and Oral Surgery, State University of Iowa Hospital; observation of the university's comprehensive speech pathology program; and special pictures and demonstrations by Prof. John S. Steinberg, of the Bell Telephone Laboratories, and Mrs. Bernice Rutherford, of the Rehabilitation Center, St. Paul, Minn.

One of the Iowa Hearing Clinics, involving medical, psychological, speech, audiometric and social work approaches to cases assembled by the Department of Otolaryngology and Oral Surgery, the Iowa State School for the Deaf, and the Speech Clinic will be scheduled during the intensive course.

For further information address Prof. Wendell Johnson, Director, Speech Clinic, State University of Iowa, Iowa City, Iowa.

THE INCIDENCE (AND DEGREES) OF BLUE SCLERAS IN OTOSCLEROSIS AND OTHER EAR DISORDERS.*

EDMUND PRINCE FOWLER, M.D.,
New York, N. Y.

Attention was called to blue scleras over 100 years ago,[1] and multiple fractures were sometimes noted in connection therewith. Later it was demonstrated that these fractures were occasioned by osteogenesis imperfecta (fragile bones) which histologically shows a resemblance to immature or embryonic bone. The combination of blue scleras with multiple fractures showed marked inheritance characteristics as a Mendelian dominant, and was frequently associated with a late progressive deafness (supposedly otosclerosis). No continuing effort was made to study the possibilities of these observations in relation to the etiology or progressiveness of otosclerosis or other osteo dystrophies.

Eddows[2] found in four generations of a family that 50 per cent of the offspring of the heterozygotic parents had blue scleras and brittle bones, and that the unaffected offspring did not have affected descendants. Only those with blue scleras had brittle bones, a generation was never skipped, and transmission was not through females to males. Bronson[3] reported a study of two families. In one (four generations), 95 per cent of those with blue scleras suffered multiple fractures and 33 per cent had deafness. In the other (three generations), 70 per cent of those with blue scleras had fractures, and none had deafness.

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Brickley[1] states that "only those members of a family who have blue scleras are subject to brittle bones and the deafness." Key,[4] in 1926, reviewed the literature, reported one case, and concluded that the tissue abnormalities seemed to be due not to faulty metabolism but to a hypoplasia of the mesenchyme, *i.e.*, an anomaly of development. Riesenman and Yater,[5] Corboy,[6] Farber and Margulis[7] and several others reported a series of patients and family groups with varying ratios of incidence. With a few exceptions no significant abnormalities in the blood calcium or phosphorus were noted.

Van der Hoeve[8] and then de Kleijn[9] and also Bronson,[3] in 1917, showed that a large proportion of cases developed an hereditary conduction deafness (otosclerosis). This deafness tended to develop in the third and fourth decades, and in advanced cases a nerve deafness was generally superimposed thereon. The obstructive deafness, because of its late development, was not noticed or was not detectable early in life. It appeared only long after the fracturing had ceased, so that even though the scleras were blue, the probability of having the triad brought to the attention of the examiner was remote. In a few instances Rutkin and others histologically demonstrated otosclerosis to be present in varying degrees, and X-rays of the petrous bone have been reported to show dense shadows, especially in the region of the vestibule. Shambaugh, in a personal communication to Brickley, reported operating upon two patients with otosclerosis and blue scleras, one with multiple fractures and the other with a daughter with multiple fractures. He found the mastoid cell walls "unusually delicate (thin) and brittle."[1] By Roentgenogram my own observations reveal that the mastoid cell walls in otosclerosis are usually thick. I have encountered only a few patients with the complete "Van der Hoeve-de Kleijn triad," but have under observation a pair of seven and one-half year old identical twins with blue sclera and fragile bones but without deafness. The parents have white scleras and normal hearing. In several instances the otological diagnosis "otosclerosis" in the cases reported in the literature with blue

scleras and brittle bones may be questioned; however, this does not qualitative alter the importance of the observations.

It is stated by Corboy[6] that about 60 per cent of the people with blue scleras suffer multiple fractures, 60 per cent have otosclerosis, and 44 per cent the complete triad. The fractures are not spontaneous, and because of the slight trauma usually involved give no great pain and show no delay in healing and tend to decrease after puberty. I infer that in such statistics reference is only to deeply blue scleras and to families with the triad, because I have observed several thousands of patients with scleras of all degrees of blueness with few multiple fractures, and many with no sign or symptom except the blue scleras to suggest the possibility of otosclerosis.

It is after puberty that the incidence and activity of the otosclerosis lesions, and the deafness therefrom, usually markedly increases, but after puberty the incidence of the fractures decreases, so that it would seem improbable that one would often observe all three of the signs of the triad simultaneously.

The sclera occupies the posterior five-sixths of the eyeball, the cornea the anterior one-sixth. In different people blue scleras may be various shades of muddy, slate, gray, clear or sky blue. In every instance I have observed the blueness to be deeper in hue in the anterior third of the sclera, and in the lesser degrees blue only in this area, the posterior third then being white. To avoid possible bias, the color of the sclera should be determined and recorded before taking the family or personal history, or making the otological examination.

The blueness is best revealed when the eye is looking downward. Some sclera appear to be clear white when the eyes are looking straight to the front, and bluish over the superior segment over the uvea when looking downward, or to the right or left. These sclera are usually marked questionable. Designating the white sclera as 0, the successive degrees of blueness are designated 1, 2, 3 and 4 plus. These are written on the history sheet as: 0, +, ++, +++, ++++.) If

there is any question as to the sclera being blue or white, it is so indicated by the question mark (?). An attempt was made to use a color scale to determine by comparison therewith the degrees of blueness, but it was found quicker and more satisfactory to dispense with it, because we could not reproduce on the scale all of the variations of blue encountered. The eyes were examined first in daylight and then by a daylight lamp.

Blue scleras are common in humans, but uncommon in the lower animals. They are associated with several disease entities involving the mesenchyme. As far as I have been able to ascertain, blue scleras when they do occur in animals are not associated with brittle bones; however, inquiries at several of our largest zoos reveal that animals do not have brittle bones. If in their natural habitat they did they would be ill equipped for life and destroyed early.

The cause of blue scleras is unknown. Some think the blueness of the sclera is due to a thinning or lessening in density of the sclera tissues so that the blue of the vitreous or uvea becomes visible. I have observed the blueness to change from time to time, but in otosclerosis I have never observed it to disappear permanently. In a few instances an increase in blueness with a coincidental increase in the deafness has been detected (not vice versa).

The blueness of the sclera is definitely influenced by excitement, emotional states, and anything which affects the circulation; premenstrual increases in blueness are common. This suggests that the capillary bed plays an important rôle, and that there may be correlations with certain endocrine tides and variations, and, therefore, with changes in metabolism in the temporal bone, and with etiologic and activating factors in otosclerosis. These possibilities excite our imagination and urge us to study the phenomena.

Table 1 shows the incidence and degrees of blueness of the sclera in 568 patients routinely examined. The patients are

TABLE 1.

INCIDENCE OF BLUE SCLERAS IN DISORDERS OF THE EARS.

Diagnosis	White Scleras			Blue Scleras			(White Patients)	
	0	?					Totals	%
	M 63	1	4	0	0		68 M	6%
	F 69	4	6	3	3		85 F	14%
	132	5	10	3	3		153	
Normal	87%	3%	6%	2%	2%			10%
	M 79	4	8	2	0		93 M	11%
	F 68	15	9	9	3		104 F	20%
	147	19	17	11	3		197	
Nerve	74%	10%	9%	5½%	1½%			16%
	M 45	1	5	2	1		54 M	14%
	F 65	11	11	13	1		101 F	25%
	110	12	16	15	2		155	
Otitis Media	71%	8%	10%	9½%	1½%			21%
	M 5	2	2	3	1	1	14 M	50%
	F 9	3	12	19	3	3	49 F	75%
	14	5	14	22	4	4	63	
Otosclerosis	24%	7%	22%	35%	6%	6%		69%
Totals	403	41	57	51	12	4	568	

divided into four categories: normal, nerve, otitis media* and otosclerosis. As in any attempt to place disease into clear-cut categories, some overlapping occurs, but for our present purpose this is of no special importance.

In studying the data one should keep in mind that probably under every category but the last (otosclerosis), as in the white population at large, 8 to 10 per cent harbored otosclerosis, although it was not diagnosable when the patient was examined, and also that the otosclerosis lesions may have been either active or inactive at the time of our examination. If any of this 8 to 10 per cent have a hidden otosclerosis, and blue scleras, as it seems reasonable to infer they have, and they were added to the otosclerosis cases it would increase the number of otosclerosis cases and the percentage of blue scleras in otosclerosis, and diminish both in the normals and in the other categories.

In several instances patients with blue scleras diagnosed as questionably otosclerotic several years ago have returned with the typical progressive deafness of otosclerosis (some were siblings). If these were added to the tabulated otosclerosis cases the incidence of the blue scleras in otosclerosis would be considerably increased. When some of these patients were seen originally their losses of hearing were only 5 to 20 db in any part of the frequency scale, there was no definite family history of otosclerosis and their personal history had been typical of slight middle ear inflammations, and, therefore, I could make only a questionable diagnosis of otosclerosis. It is extremely risky to let unproved positive or negative tests or signs influence one's judgment in diagnosis, but it does no harm and may be of value, if we routinely look for and record them for future reference. There are exceptions to many of the most widely accepted positive and negative diagnostic criteria.

The normal hearing controls, as they showed no deafness, must have possessed some and probably a definite defense

*Those with evidence of nonsuppurative or suppurative inflammatory episodes in the past or present with or without a history of same.

against the development of otosclerosis, and because we have separated this control group from the deafened people, fewer may be presumed to have harbored the lesion than in the white population at large. At any rate it is interesting to note that there are few questionable blues among the normals (*i.e.*, 3 per cent), whereas 8 per cent were questionable in otitis media, 10 per cent in nerve, and 7 per cent in otosclerotic cases. If in the normal controls the questionable cases are counted as white, the percentage of normals with blue scleras will be 10 per cent. In the normal hearing males the incidence of definitely blue scleras was 6 per cent, and in the females 14 per cent. This is identical with the incidence figure for otosclerosis in males, and within 2 per cent of the figure for females in Guild's[10] autopsy data (6 per cent and 12 per cent, respectively). It may be that instead of "incidence figure" a better word would be "coincidence figure." Coincidences, however, are facts and merit study.

The nerve cases were in some instances probably complicated by otosclerosis, although a diagnosis of otosclerosis with nerve deafness was at the time impossible or uncertain. Nevertheless, it may be noted that a greater percentage (16 per cent) showed blue scleras than in the normals. This may be due to the fact that here is a group in which we were unable to diagnose with certainty several who also had otosclerosis deafness. The percentage in males and females was 11 per cent and 20 per cent, respectively.

The otitis media cases with few exceptions showed obstructive deafness and here the diagnosis of otosclerosis is more difficult to make than in the foregoing category. It is a common practice to shy away from making a diagnosis of otosclerosis in a suppurative ear. But otitis media and otosclerosis may, and often do, occur together. (See Guild's data.[10]) The percentage of blue scleras (21 per cent) is greater than in the previously discussed group, and twice that in the normals. The percentage in the males and females was 14 per cent and 25 per cent, respectively. It is possible that this increased incidence of blue scleras may indicate that there is

a larger percentage of otosclerosis in otitis media cases than we have heretofore been led to believe obtains. Much more data is required for proof of such an assumption.

In the otosclerosis cases there is a dramatic increase in the incidence of blue scleras (69 per cent); seven times that in the normals. This, in view of the number examined, is too great for mere coincidence. The percentage in males and females was 50 per cent and 76 per cent, respectively.

Brown eyes and blue eyes, like otosclerosis, show strong hereditary backgrounds, and the geneticists have established theories concerning the inheritance factors involved. Blue scleras also show hereditary traits. In several families I have noticed that the siblings with blue scleras developed otosclerosis and those with white scleras did not. (There are exceptions.) In one set of identical twins, when originally examined the twin with blue scleras had developed otosclerosis deafness and the twin with white (or questnonably white) scleras had not. Two months after her first pregnancy this latter twin developed two plus blue scleras. The child of this twin has two plus blue scleras. At birth they were a much darker blue. The mother is nursing the baby. As yet she shows no significant changes in her hearing.

Women evidently are exposed to greater activating factors than men. The ratio of otosclerosis in white women and men observed histologically is about two to one, respectively; observed clinically it appears to be three or more to one. One reason for this seeming discrepancy may be that at autopsy we discover both the small and large, the early and late lesions; whereas clinically we diagnose only the large or well advanced lesions. Otosclerotics do not come to us before the onset of deafness, and after deafness occurs the lesion is large and far advanced.

Another reason is that otosclerotic patients are usually very healthful, they may not so often suffer and die from the diseases that usually demand an autopsy as in the population at large; moreover, on the average otosclerosis deafness appears

earlier and advances more rapidly in women than in men, so that we see many more young women than young men with otosclerosis. I have elsewhere explained how this may happen.[11] In this connection it should be pointed out that in Guild's data between the ages of 30 and 50 the ratido of males to females with otosclerosis is one to two, but before 30 it is one to three or more, the same as we observe it clinically. It may be, and it is my impression, that the young women are more prone to consult the otologist early than are the young men, at any rate in every category in the table the female patients outnumbered the males.

Assuming that in my own as in your patients we examine a fair sample of the white population (with and without deafness), it would be logical to expect that if more women than men suffer from otosclerosis we will see more women than men in our offices, and by and large these women and men will harbor otosclerosis in about the same ratio as it occurs in women and men in the general population, but we do not see a fair sample; we see a larger proportion of people with deafness and a considerable portion of this deafness is due to otosclerosis. At any rate this is the way it seems to me.

It should be especially noted that the incidence of otosclerosis increased noticeably with the overall intensities of the blueness of the scleras; 3 per cent of the white scleras; 12 per cent of the questionably blue scleras; 35 per cent of the total number of blue scleras; 45 per cent of the two, three and four plus blue scleras; 50 percent of the three and four plus blue scleras, and 100 per cent of the people with four plus blue scleras were definitely diagnosed "otosclerotics."

These findings are what should be expected if there is a definite relationship between blue scleras and otosclerosis.

In every category the incidence of blue scleras was higher in the females than in the males, and with the exception of otosclerosis it was about twice as great in the females as in the males. In otosclerosis the ratio was three to two. If there is some etiologic factor common to blue scleras and otosclero-

sis, these ratios are what should have been expected. If they had not obtained, it would have mitigated against any close relationship between the two. Contrawise, because of the rare occurrence of brittle bones in otosclerosis we may infer that there may be no close inter-relationship between the factors underlying otosclerosis and osteogeneses imperfecta. Not until a large number of cases of the latter are available for study will it be possible to determine whether the incidence of otosclerosis in osteogenesis imperfecta is greater than in the blue sclera population at large, although the incidence of blue scleras appears to be high in both conditions.

SUMMARY AND CONCLUSIONS.

A brief reveiew and discussion of the literature has been set forth. Five hundred sixty-eight patients first examined as to the bluess of their sclera, and then otologically, are tabulated in four categories (normal, nerve, otitis media and otosclerosis), and under six degrees of blueness of the scleras.

The incidence of blue scleras in those with otosclerosis is 69 per cent, as compared with 10 per cent in normal hearing controls, 16 per cent in nerves, and 21 per cent in otitis media deafness. These percentages and the ratios of males to females are discussed and several significant agreements and disagreements with clinical and autopsy findings are pointed out.

Otosclerosis was diagnosed in 3 per cent of those with white scleras; 12 per cent of those with questionably blue; 35 per cent of those with two or more degree of blueness; in 50 per cent of those with three or more degrees of blueness; and in 100 per cent of those with four degrees of blueness of the sclera.

These, and the other findings, coincide so closely with what should be expected if there is a definite relationship between blue scleras and otosclerosis that they should not be ignored. A larger sampling and continuing studies will determine more clearly (especially in families with a positive history of otosclerosis) the importance of the color of the sclera for etilogi-

cal investigations, and for a preclinical or prognostic diagnosis of otosclerosis, and above all let us hope for preventive treatment.

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140 East 54th Street.

THE FUTURE OF OUR SPECIALTY.*

HOWARD MCCART, M.D.,
Toronto, Canada.

I express my gratitude for the honor you have bestowed upon me, and ask your continued support of this Society. This can be accomplished in several ways, among which is active membership in your Society and your participation in the free discussion of the papers on the program. The success of any society depends on the enthusiasm of the Fellows and the conscientious effort of the officers to carry out your wishes. A large membership does not necessarily make for the success of a society, but I am of the opinion we should endeavor to expand the membership of the Canadian Otolaryngological Society, providing each member is willing to play an active part. The late Dr. James Babbitt, in his presidential address before the Academy of Ophthalmology and Otolaryngology, stated that he was aware of two groups in our specialty — those within the Academy, and those without. To those without our Society, may I ask for your membership, which will be greatly advantageous to all of us in the everchanging days to come, by guarding majority rather than the individual rule? Your support of the Society may at times seem intangible, but by so doing, you can play your part in the broader field of Canadian medicine, thus assuring leadership in our specialty and assisting in the training of otolaryngologists in our country.

There has always been a close relationship between ophthalmology and otolaryngology. Many of our Fellows are mem-

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bers of the ten-year-old Canadian Ophthalmological Society. As many of you practice both specialties, it is only fitting that a closer association should be fostered between the two societies.

As the name of our specialty implies, it is Diseases of the Ear and Larynx, and includes Diseases of the Throat, Pharynx, Nose and Nasal Sinuses, Trachea, Bronchi and Esopahgus. It is evident that this somewhat imposing array of diseases will not remain in our specialty permanently, unless we are prepared to broaden our knowledge and training.

It is interesting to review briefly the history of otolaryngology. We are young in many ways. It was in 1839 that James Yearsley adopted otology as a specialty and founded the Metropolitan Ear Hospital in London, Wilde, about 1841, was the first to teach otology in the United Kingdom, the students coming from all over the world, especially America. Hinton, in 1868, was probably the first surgeon in Britain to operate on mastoiditis, which is somewhat ironical, as at the present time, 80 years later, acute mastoid surgery is rare. Meyer (1868), in Copenhagen, discovered adenoids. Joseph Toynbee, the great pioneer otologist, placed otology on a sound basis: he was the first to describe otosclerosis, mentioning a report of 160 cases in the year 1860. In 1859, only 89 years ago, Marshall Hall stated, in the *Lancet*, that we must regard tracheotomy as a heroic remedy only appropriate to Herculean forms of disease. Sir Charles Ballance (1856-1936), another pioneer, was noted for his work on mastoiditis and facial paralysis; and lastly, Sir William McEwen (1848-1924), the results of whose operations for otogenic cerebral abscess are still remarkable.

Previously, ophthalmology and otology were practiced together. Laryngology came into being as a branch of general medicine rather than an offshoot of surgery; nor did it advance as a special branch of medicine until Garcia, in 1854, discovered the laryngeal mirror. Morrell McKenzie (1837-1892) should be regarded as the founder of British laryngology. Sir Patrick Heron Watson (1832-1907) performed the first laryngectomy, and Sir Felix Semon (1849-1921) was

one of the last to practice laryngology as an independent specialty. Early in the present century otology merged with laryngology to become otolaryngology.

There are many of us who, for some years, have felt that there is something lacking in our specialty, that it has changed, that it is becoming narrower; perhaps it is that we, the specialists, have narrowed. After World War I, many men without adequate training were attracted to ear, nose and throat practice, largely because of the trend in medicine to search for a focus of infection in all systemic diseases. In many cases, and often at the request of the internist, possible foci were removed in arthritis, bronchitis, rheumatic fever, etc., and the results found wanting. Today there is less unnecessary surgery, fewer sinuses punctured, fewer Eustachian tubes catheterized. We have a better understanding of the nasal mucosa and its function, much of which can be credited to Proetz. We have more respect for the nasal mucosa of an allergic patient, though we understand it less. Perhaps, too, our knowledge or understanding of psychosomatic medicine has improved, thus recognizing that a patient's emotions may throw him off balance, affecting remote organs. Wolff and his co-workers have shown reactions of emotional stress in relation to the mucous membrane of the nose, one in which shrinking of the nasal mucosa occurred, accompanied by the emotion of fear and sadness, and again initial hyperemia of turbinates, septum and nasal mucosa, with resultant nasal obstruction occurring with anger and feeling of frustration. Thus emotional states may often require study in chronic sinusitis, vasomotor rhinitis and headache, or may be an underlying factor in the common cold. Lastly, our specialty has changed or narrowed, due to chemotherapy and antibiotics.

Recognizing these changes, we must improve on our predecessors' methods and intentions. It is my belief that this is an opportune time for us as specialists to reflect and lay plans, rather than to wait until inroads are made in our specialty. A recent inroad appears to be in bronchoscopy.

Bronchoscopy in most centers has always been, and rightly so, a branch of otolaryngology. Our weakness lay in that all

of us did not include it in our specialty, due in some cases to lack of interest or the belief that there was not an opportunity to do this work in our centers — to those, I say that you would be surprised if you would attach yourself to sanataria, with or without recompense. This is one means of broadening your specialty. As a result of this lack of interest in some centers, chest physicians and surgeons have recently undertaken this work. I do not object to any physician or surgeon doing bronchoscopy, providing he is adequately trained, but then only in centers where ear, nose and throat service is not provided. Nor is the medical or surgical problem always solved by bronchoscopy, but rather does the case need further clinial investigation. Bronchoscopy is not a field for everyone. The examination of a few cases will not enable one to become skillful or able to recognize the variety of conditions that may be encountered. In larger centers, especially in university centers, it should be carried out by the department of otolaryngology, as it is only by such means that our future otolaryngologists can be trained in this work, or become deft in the removal of foreign bodies from the bronchi and esophagus, and become quickly familiar with the different diseases of these organs. Otolaryngologists are more eminently suited for this work, as they are used to working in small fields and have developed monocular vision.

Not too many years ago, otolaryngologists did thyroid surgery. I do not see why this again should not be included in our specialty, also deep neck, meningeal and major plastic surgery. Brain abscesses were formerly in the field of otolgy. The modern otologist should have a working knowledge of neurology. He should be competent to open a cerebellar abscess, either by the internal route, *i.e.*, mastoid, or by the external route. It is only a step in surgical technique to perform block dissection on the glands of the neck, or to remove esophageal diverticula for one trained to perform major laryngeal surgery.

More otolaryngologists must become qualified to deal with cancer of the larynx. The surgical treatment over a period of years will show a high percentage of cures, especially in the

intrinsic cases. I also believe that unless the laryngologist is prepared to carry out the operation himself, he is better advised to let the operating surgeon do the biopsy; if not, it is often difficult for him to determine the extent of the lesion, especially if it is near one or the other commissure.

How shall we best improve on our predecessors' methods and intentions, feeling that otolaryngology is perhaps at a crossroad? The present training for otolaryngologists in Canada consists of two years, including the basic sciences, preceded by a general intern year. Thus we have established certain standards for training and defined them. Some of you are of the opinion this time is too long and unnecessary. This is not true, as surgery and its specialties, more than other divisions of medicine, demand intensive and prolonged training.

Certification is a means of eliminating the self-styled specialist and giving a diploma to those successful in passing this examination. I wonder if it would not be much better for our specialty to have a Fellowship examination, following the plan of surgery, obstetrics and gynecology, and genitourinary surgery.

If such a plan was followed, a student contemplating the study of otolaryngology, upon completion of his general intern year, would be required to take one year of general surgery, then should follow a period of training of not less than two years in otolaryngology, during which time instruction in the basic sciences could be given within the medical school. The added year in general surgery and basic sciences should greatly aid otolaryngologists to become more skillful surgeons, enabling them to try the Fellowship examination and to be competent to perform all surgery of the head and neck.

The problem of the deaf is a serious health problem in Canada. While no survey has been made in Canada, except in isolated districts, it would not be unfair to accept the figure of the United States, which is that 6 to 7 per cent of the population has a hearing impairment. This Society should take concerted action regarding this problem and a committee

formed from each province, presenting their views before their provincial minister of health, so that we can prevent and ameliorate hearing defects and rehabilitate those persons who have lost serviceable hearing.

The care of the deaf must be attacked early in life and should be a conjoined program between the departments of health and education. We must draw the government's attention to the fact that we can diagnose and identify those individuals who are hard of hearing, that we have in many cases adequate therapeutic measures for those that show promise of a favorable prognosis, and that they, the government, must provide the buildings, equipment and teachers to rehabilitate those individuals who require special education by remedial instruction in speech reading, voice correction and vocational guidance.

In some provinces, facilities are available in a limited manner for rehabilitation of those without serviceable hearing. These institutions, if or when they are located, need many teachers of speech reading. Speech reading is absolutely essential for normal social relationship. In obstructive deafness, hearing aids give a return of this normal relationship, but when progressive deafness reaches a profound state, unless the patient has received speech reading early, he has nothing to fall back on in order to maintain this social relationship. These institutions should be located at university centers, as it is only at such centers that many of the very necessary affiliated departments such as speech, physiology, psychology, physics and otolaryngology are available.

Leadership in medicine and our specialty is essential. So far, in this country it has been allowed to develop freely and independently. How long this freedom will continue is difficult to say. The various health insurance schemes in force or contemplated may curtail this freedom. It is only by strong leadership in our profession that the high quality of medical education and medical services can be maintained.

If this leadership becomes weak, we leave a poor heritage for those who follow, attracting youth without the necessary

culture or vision, with the inevitable result of lowering our standard of medical education and the relationship between the patient and the physician. Our country was founded by pioneers seeking freedom, and of such stock the medical profession of the North American Continent rose to its present heights without governmental regulations. I am not unmindful of the excellent work that can be done by governmental health departments — such as in the treatment of tuberculosis, mental hygiene, rehabilitation of the blind and, we hope before long, a modern institution for the deaf. Many of these people are handicapped for life and need institutional care, the upkeep of which must be borne by the state. Many, and perhaps all, would be better for individual physician care were it not due to the prohibitive cost; however, I fail to see how complete governmental control of all medicine can offer the patient a higher standard of medical service. It might rise in a few outlying districts but, on the whole, would fall in due course, finally attracting the type of student with intellectual inertia, interested only in his personal social security.

I do not feel that anyone here will hold any brief with me when I say that medical leadership today is on the North American Continent and, as members of such, we must take a broader part and accept greater responsibility in postgraduate education of our Canadian otolaryngologists. This leadership became necessarily more pronounced at the conclusion of the late war. Canadian postgraduate teaching now has to rely on our own universities and teaching hospitals for leadership and instruction, because at the end of the war, many of our graduates were unable to pursue their postgraduate study, which had formerly been taken in Great Britain and the United States, as these countries were faced with a return to civilian life of a large number of their physicians seeking postgraduate instruction.

This places a heavy and extra burden in many instances on the university and hospital staffs, but not without its reward, as I feel certain the efficiency of a staff will improve when they have to be responsible for the fundamental training in a specialty. Such being the case, this Society should appoint a

committee on otolaryngological teaching in Canada, so that we may rate our universities and hospitals, rather than be dependent upon the classification placed by the Rockefeller Foundation. If such a committee is formed from this Society, I feel you will more likely have an aggressive Society. Failing this, no matter how lofty the ideals of a Society may be, and our ideals are laid down in our constitution, we will soon become a Society where nothing is accomplished except the presentation of papers; we will be short lived or, even worse, develop or be governed by a clique.

We, in Canada, should feel fortunate that we can accept the responsibility of postgraduate teaching in otolaryngology; fortunate that none of our great teaching institutions were damaged in the recent world conflict. Travel and education previous to the last war helped many of our physicians and surgeons to consolidate the best teaching from abroad and at home. May the Canadian profession continue by tradition to automatically pick out the best from our conservative British forefathers and the best from the advanced ideas of our neighbors to the south.

I believe in the future of otolaryngology and have drawn to your attention some of the points necessary to insure the continuation of a sound and broad specialty. This can be brought about only by increasing our general surgical training in order that we may be competent to carry out surgery of the head and neck. Leadership must come through this Society if otolaryngology as a specialty is to hold its rightful place in the practice of medicine. Let the Fellows of the Society break the bonds of narrow satisfaction, courageously attacking our responsibilities with wisdom and foresight, so that we may become broader medical men and better surgeons.

ESOPHAGEAL VARICES AND THEIR TREATMENT BY INJECTION.*

JOHN F. TOLAN, M.D.,
Seattle, Wash.

The treatment of esophageal varices by injection of a sclerosing solution into the veins through an esophagoscope has been a source of considerable interest to esophagologists. The first real work in this field carried out in this country was done by Dr. Herman Moersch, of the Mayo Clinic. My interest in this particular procedure was not only stimulated by his apparent success but also by several tragic experiences which he had at our clinic, caused by varices of the esophagus.

In order to better understand the rationale for treating varices in the esophagus by injection of sclerosing solution it is advisable to review briefly some of the mechanisms involved in their production. The etiology underlying the development of esophageal varices is not entirely known. There are two theories generally advanced to account for their development: first, those who have expressed the opinion that the primary difficulty is caused by disturbance of the vascular regulatory apparatus of the spleen so that the arterial blood flows unobstructed into the venous system and produces a tract analogous to that of an arterial venous fistula. On the other hand, there are those who postulate that the difficulty arises as a result of obstruction in the splenic or portal vein owing to either intrinsic or extrinsic causes, producing an obstruction to the free passage of blood back to the systemic

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circulation. This gives rise to a state of portal or splenic hypertension. In either case there occurs marked engorgement of the portal circulation owing to the inability of the splenic or portal vein to return the blood adequately to the systemic circulation.

Nature tends to compensate for this difficulty and to establish new channels to return this stagnant blood to the general circulation. This attempt as a rule generally takes place through the channels of communication that already exist between the portal and the systemic circulation. The anastomosis that most frequently results and with which we are most concerned is that which leads to the development of the esophageal varices. This anastomosis, as McIndoe has pointed out, is by way of the coronary vein, which likewise is the most direct means of communication between the portal and the systemic circulation. The coronary vein of the stomach branches as it pierces the muscular coat to reach the submucosa of the cardia, where it divides into a number of small longitudinal medials which have no cross-communication for 3 or 4 cm. At this level they break up into a rich plexus which anastomoses freely, and which may extend into the upper third of the esophagus. The communicating branches run from the plexus to large periesophageal trunks which, in turn, communicate with the intercostal branches through the diaphragmatic vein to the systemic circulation. The veins of the submucosa of the esophagus are very poorly supported by loose connective tissue.

As a result, varices develop because the increased flow of blood through the veins and the inability of the muscular elastic fibres to withstand the increased venous pressure. In addition, the tendency towards the formation of varicosities is accentuated by the effects of increased intrathoracic pressure.

Esophageal varices, as seen through the esophagoscope, present a characteristic picture. The varicoceles appear as tortuous, often nodular, light bluish prominences which protrude into the esophageal lumen; they are easily compressible and change shape with peristalsis of the esophagus. They

tend to run in a longitudinal plane and may be so large and numerous that they occupy the entire esophageal lumen.

The diagnosis of the esophageal varices is too often made at the autopsy table. One should be suspicious in any case where there is a history of unexplained massive gastrointestinal bleeding.

Careful Roentgenological examination of the esophagus will usually reveal the presence of varices. Occasionally the diagnosis can be made only by esophagoscopy.

The purpose of injecting a sclerosing solution into the esophageal varices is to block the free passage of blood through the esophageal vein. One hopes to stimulate a border circulation to increase its efficiency in conveying blood back through the liver to the systemic circulation in a normal manner; however, the more important reason is the stimulation of new anastomotic channels to convey the blood from the portal to the systemic circulation by such a route that the communication will not be subjected to trauma as occurs in the esophagus.

Splenectomy, as we know, is the procedure which has given the most satisfactory results in cases in which the esophageal varices were due to splenic anemia. Splenectomy alone will cure only that relatively small group of thrombosis of the splenic veins in which the site of obstruction is distal to the junction of the coronary veins. Dr. Will Mayo pointed out that this procedure should be of value if for no other reason than that the removal of the spleen decreases by 20 per cent the amount of blood that passes through the portal circulation. In so doing, this lessens the amount of blood that must be transmitted back to the systemic circulation by the portal system. Recurrence of hemorrhage subsequent to splenectomy is, however, not unusual. The recurrence of hemorrhage from esophageal varices after splenectomy takes place in approximately 50 per cent of the cases.

It becomes readily apparent, therefore, that any procedure that can prevent such bleeding is deserving of serious consideration. In 1933, Pemberton and Walters each suggested

the advisibility of injecting a sclerosing solution into the esophageal varices. They suggested injection through an esophagoscope or into the coronary vein at the time of surgical intervention. Crafoord and Franckner, of Stockholm, in 1939, were the first to successfully inject the esophageal varices with a sclerosing solution through an esophagoscope. Their success was shortly followed by Moersch's work in this country.

The apparatus for injection consists of a firm 11 gauge tube. To this a 24 calibre hypodermic needle is attached. The proximal end of the tube is bent at right angle to permit free visualization. At the proximal end of the tube a short piece of rubber tubing is fitted to allow the attachment of the syringe containing the sclerosing solution. The use of the rubber connection is of great value in preventing unnecessary jiggling of the needle during the injection. A removable handle which can be attached to the proximal end of the needle has been found of considerable assistance in guiding the needle properly and steadily.

The solution which was used by Crafoord and Ranckner was quinine hydrochloride. Moersch instigated the use of sodium morrhuate because it is less likely to produce sloughing and severe systemic reaction. He first used a 2.5 per cent solution of sodium morrhuate; at the present time a 5 per cent solution is being used because it seems to produce somewhat better and more rapid sclerosis.

The technique used, of course, is first the passage of the esophagoscope to the desired level, and a suitable vein is selected for injection. The scope is placed gently against the varix, just proximal to the point at which the needle is to be inserted. The needle then is inserted into the vein. We find it advisable, when in doubt, to remove some blood from the lumen of the vein to make sure that the needle is in its proper place. With the needle in proper position, the solution of sodium morrhuate is injected slowly into the vein. The amount of solution used depends upon the patient's reaction. I have injected as much as 9 cc. at one treatment without any ill effect; however, it is advisable to limit the amount

injected on the first treatment to be certain that the patient is not sensitive to the drug. Only one vein is injected at a time. When the needle is withdrawn, there invariably occurs at the site of puncture a small amount of bleeding which can easily be controlled by adrenalin or thromboplastic tampons.

In some cases the patient complains about pain which is usually substernal and extends through to the back. If pain occurs, it usually takes place after 1 cc. to 2 cc. of the solution is injected. The solution may be escaping into the perivascular tissues, or may be due to a spasm in the vessel itself. The pain, however, disappears in 12 to 24 hours. Subsequent changes in the vessels take place and they take on a firm, small, pale appearance.

1. Hemorrhage or ascites, or both, can occur in cirrhosis cases purely on the basis of depressed liver function. *2.* Hemorrhage may be due to so-called wasting ascites caused by portal hypertension and at the same time have a perfectly well compensated liver. *3.* Hemorrhage may be due to the inability of the liver to produce albumin or prothrombin. It is of utmost importance to improve liver function to its maximum before making one's decision on the operative procedure.

In my series of 19 cases, the ages ranged from 14 to 58. There were 10 males and nine females. Twelve cases were so-called typical Banti's, and in seven of these cases varices was diagnosed before splenectomy. In the remaining seven cases of the series, the diagnosis of intrahepatic hypertension, or cirrhosis, was made. Three of these patients had poor liver function with inability to produce sufficient prothrombin and albumin. All of the splenectomy cases suffered hemorrhages before operation.

Four of the splenectomized cases went for a period of two to five years before they had a recurrence of bleeding. The remaining cases bled anywhere from one month to 18 months after splenectomy. *The three cases of poor liver function did very poorly.* One of the cases of intrahepatic hypertension with extremely good liver function has gone 16 months, during which time he has been injected on four occasions. The

remaining three in this group are more recent. In the 12 cases of Banti's there were three deaths. One of these had gone a little over three years, during which time the varices were injected on five occasions. One of the cases has gone five years since I injected him last. This man is a secretary for one of our senators from Washington and he informed me that on only one occasion did he have any suggestion of gastrointestinal bleeding.

The remaining cases ranged from six months to 18 months. In two of these cases I have been unsuccessful in controlling completely their gastrointestinal hemorrhages, but they are greatly improved. The other two cases are doing very nicely at the present writing, and one has gone eight months since the last injection, and the other, 13 months.

The blood loss at the time of gastrointestinal hemorrhage in some amounts to as much as one to two liters at each episode. In a few of the cases the hemoglobin dropped as low as 40 per cent. The greatest number of injections in any one case was 12. The total amount of solution used in any one case was 28 cc.

Blakemore, of New York, is doing extensive work on the portocaval anastomosis and also the anastomosis of the splenic and renal vessels. In his series of 58 cases he has had 11 postoperative deaths; however, I do feel that insofar as vascular surgery is concerned, he has opened up new hopes for further treatment of these cases.

SUMMARY.

1. Splenectomy alone, although the most reliable and time honored surgical treatment for this condition, is not the conclusive answer to the problem.

2. Splenectomy by relieving over 20 per cent of the load from the portal circulation, followed by the injection of a sclerosing solution into the varices, has proved very helpful. These injections, however, have had to be repeated in many cases.

3. Blakemore's portocaval anastomosis, combined with the above procedures, will add greatly to the success in treatment of esophageal varices.

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"THE FUNCTION OF THE EAR IN HEALTH AND DISEASE."

Unusual Educational Sound and Color Film, Edited by H. G. Kobrak, M.D., Ph.D.; Joseph E. Hind and Robert B. Miller, M.D.

This film was produced at the University of Chicago and its purpose is to demonstrate the response of the ear to sound in health and disease. The range which the ear can perceive is demonstrated. The minimum and maximum intensity of sound recorded by the ear is shown. Pathology of the middle ear is demonstrated on diseased temporal bones. The vibrating eardrum after paracentesis is demonstrated. The inflation after Politzer and the effect on the eardrum is shown.

Hearing curves of certain types of deafness are shown and the response of the ear is demonstrated. By using electronic filters, the sound perception of a diseased ear is given in the sound track similar to the actual hearing defect; therefore, the audience is able to see the pathologic function on the screen and simultaneously hear the distorted acoustic signal which the patient perceives.

The audiometric hearing curve of a patient obtained before fatal termination of his disease is shown. The acoustic vibrations of this diseased temporal bone are demonstrated.

The action of hearing aids and intensity changes due to the action of the hearing aid is demonstrated. The reflex of the middle ear muscles is demonstrated in animal experiments and in patients. The utilization of the stapedius reflex for objective hearing tests is demonstrated.

This is the first film of its kind ever attempted and successfully accomplished for visual education in otology.

The film is available to medical societies, hospitals, universities and professional study groups without charge through the Auralgan Research Division, 100 Varick Street, New York 13, N. Y.

MISSISSIPPI VALLEY MEDICAL SOCIETY
1949 ESSAY CONTEST.

The Ninth Annual Essay Contest of the Mississippi Valley Medical Society will be held in 1949. The Society will offer a cash prize of $100.00, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association who are residents and citizens of the United States. The winner will be invited to present his contribution before the Fourteenth Annual Meeting of the Mississippi Valley Medical Society to be held in St. Louis, Mo., Sept. 28, 29, 30, 1949, the Society reserving the exclusive right to first publish the essay in its official publication — the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). All contributions shall be typewritten in English in manuscript form, submitted in five copies, not to exceed 5,000 words, and must be received not later than May 1, 1949. The winning essays in the 1948 contest appear in the January, 1949, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.).

Further details may be secured from Dr. Harold Swanberg, Secretary, Mississippi Valley Medical Society, 209-224 W. U. U. Building, Quincy, Ill.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1949.

Acousticon Model A-100.
Manufacturer: Dictograph Products Corp., 580 Fifth Ave., New York 19, N. Y.

Aurex (Semi-Portable); Aurex Model C-B, Model C-A, Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver).
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model A-1; Otarion Model A-3; Otarion Models A-4 J and S; Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Precision Table Hearing Aid.

Manufacturer: Precision Electronics Co., 850 W. Oakdale, Chicago 14, Ill.

Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Ravox (Semi-Portable).

Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C.

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Telex Model 22; Telex Model 97; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A."

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vactuphone Model 3.

Manufacturer: Allen-Howe Electronics Corp., Salem, Mass.

Western Electric Orthotronic Model; Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

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